

Contributors

CONSULTING EDITOR

RICHARD H. HAUG, DDS

Carolinas Center for Oral Health, Charlotte, North Carolina

EDITOR

DAVID A. BITONTI, DMD, CAPT, DC, USN

Senior Military Advisor to the Commander, Walter Reed National Military Medical Center, Bethesda, Maryland

AUTHORS

ROCCO A. ARMONDA, MD

Director, Cerebrovascular Surgery and Interventional Neuroradiology, Division of Neurosurgery, Walter Reed National Military Medical Center, Bethesda, Maryland

RANDY S. BELL, MD

Division of Neurosurgery, Walter Reed National Military Medical Center, Bethesda, Maryland

JEFFREY P. BLICE, MD, Capt, MC, USN

Ophthalmology, Walter Reed National Military Medical Center; Assistant Professor of Surgery, Uniformed Services University of Health Sciences, Bethesda, Maryland

WILLIAM J. BURKE, DMD

Department of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center; Resident, Oral and Maxillofacial Surgery Residency Program, National Capital Consortium, Bethesda, Maryland

RAMON F. CESTERO, MD, FACS

Commander, Medical Corps, Naval Medical Research Unit, United States Navy, Fort Sam Houston, San Antonio, Texas

RODNEY K. CHAN, MD

U.S. Army Institute of Surgical Research, Fort Sam Houston, Texas

CHRIS CRECELIUS, DDS

Division of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center, Bethesda, Maryland

ROBERT I. DELO, DDS, MD

Colonel (Ret), US Air Force Medical Service, Consultant to the US Air Force Surgeon, General for Oral and Maxillofacial Surgery, Department of Oral and Maxillofacial Surgery, Lackland AFB, Texas

STEVEN V. DRYDEN, DDS

Staff Surgeon, Division of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center, Bethesda, Maryland

ISAAC D. ERBELE, MD

Department of Otolaryngology Head and Neck Surgery, Walter Reed National Military Medical Center, Bethesda, Maryland

MICHAEL A. GENTILE, DMD

Staff Surgeon, Department of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center; Associate Program Director, Oral and Maxillofacial Surgery Residency Program, National Capital Consortium; Assistant Professor, Department of Surgery, Uniformed Services University of Health Sciences, Bethesda, Maryland

GERALD T. GRANT, DMD, MS, Capt, DC, USN

Service Chief, Department of Radiology, 3D Medical Applications Center, Walter Reed National Military Medical Center; Director, Craniofacial Imaging Research, Navy Medical Personnel Training Center, Naval Postgraduate Dental School NMPTC, Bethesda, Maryland

ROBERT G. HALE, DDS

Commander, Division of Dental and Trauma Research, U.S. Army Institute of Surgical Research, Fort Sam Houston, Texas

CHRISTOPHER M. HARRIS, DMD, MD, LCDR, DC, USN

Residency Program Director, Oral and Maxillofacial Surgery, Naval Medical Center Portsmouth, Portsmouth, Virginia

VISIT...

LANZAROTE
Caliente.COM

Contributors

MICHAEL S. JASKOLKA, MD, DDS

First Appalachian Craniofacial Deformity Specialists, Multi-Disciplinary Cleft and Craniofacial Disorders Clinic, Women and Children's Hospital, Charleston Area Medical Center; Charleston Division, Department of Surgery, West Virginia University, Charleston, West Virginia; Adjunct Clinical Instructor, Department of Oral and Maxillofacial Surgery, University of North Carolina, Chapel Hill, North Carolina

JAE H. KIM, DDS

Resident, Oral and Maxillofacial Surgery Residency Program, National Capital Consortium, Division of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center, Bethesda, Maryland

SHAYNE KONDOR, BAE, MAE

Contractor, Craniofacial Imaging Research, Navy Medical Personnel Training Center, Naval Postgraduate Dental School, Bethesda, Maryland

ROBERT LAUGHLIN, DMD, LCDR, DC, USN

Residency Program Director, Oral and Maxillofacial Surgery, Naval Medical Center San Diego, San Diego, California

PETER LIACOURAS, PhD

Director of Services, Department of Radiology, 3D Medical Applications Center, Walter Reed National Military Medical Center, Bethesda, Maryland

DAVID B. POWERS, DMD, MD, FACS, FRCS (Ed)

Associate Professor of Surgery, Director, Duke Craniomaxillofacial Trauma Program, Division of Plastic, Maxillofacial and Oral Surgery, Duke University Medical Center, Durham, North Carolina

J. MICHAEL RAY, DDS

Assistant Professor, Department of Oral and Maxillofacial Surgery, Baylor College of Dentistry, Dallas, Texas

ARNALDO RIVERA, MD

Department of Otolaryngology Head and Neck Surgery, Walter Reed National Military Medical Center, Bethesda, Maryland

MERYL A. SEVERSON III, MD

Division of Neurosurgery, Walter Reed National Military Medical Center, Bethesda, Maryland

WILLIAM G. SHOEMAKER, DDS

Service Chief, Division of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center, Bethesda, Maryland

M. PETER SORENSEN, MD

Department of Otolaryngology Head and Neck Surgery, Walter Reed National Military Medical Center, Bethesda, Maryland

ANDREW J. TELLINGTON, DDS

Department of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center; Resident, Oral and Maxillofacial Surgery Residency Program, National Capital Consortium, Bethesda, Maryland

DAVID I. TUCKER, DDS

Chief of Oral and Maxillofacial Surgery, Division of Dental and Trauma Research, U.S. Army Institute of Surgical Research, Fort Sam Houston, Texas

MICHAEL R. ZACHAR, DDS

Chief Resident of Oral and Maxillofacial Surgery, San Antonio Military Medical Center; Department of Oral and Maxillofacial Surgery, Brooke Army Medical Center, Fort Sam Houston, Texas

Contents

Preface: Craniomaxillofacial Trauma	vii
David A. Bitonti	
Initial Management of the Trauma Patient	1
J. Michael Ray and Ramon F. Cestero	
The Treatment of Maxillofacial Trauma in Austere Conditions	9
J. Michael Ray	
Characteristics of Ballistic and Blast Injuries	15
David B. Powers and Robert I. Delo	
Maxillofacial Imaging in the Trauma Patient	25
Gerald T. Grant, Peter Liacouras, and Shayne Kondor	
Wound Management and Nutrition for Optimal Wound Healing	37
Steven V. Dryden, William G. Shoemaker, and Jae H. Kim	
Soft Tissue Trauma	49
Chris Crecelius	
Characterization and Management of Mandibular Fractures: Lessons Learned from Iraq and Afghanistan	61
David I. Tucker, Michael R. Zachar, Rodney K. Chan, and Robert G. Hale	
Management of Midface Maxillofacial Trauma	69
Michael A. Gentile, Andrew J. Tellington, William J. Burke, and Michael S. Jaskolka	
Ocular Injuries, Triage, and Management in Maxillofacial Trauma	97
Jeffrey P. Blice	
Triage and Management of Cranial Injuries	105
Meryl A. Severson III, Randy S. Bell, and Rocco A. Armonda	
Otologic and Temporal Bone Injuries, Triage, and Management	117
Isaac D. Erbele, M. Peter Sorensen, and Arnaldo Rivera	
Reconstruction of Hard and Soft Tissue Maxillofacial Defects	127
Christopher M. Harris and Robert Laughlin	

ATLAS OF THE ORAL AND MAXILLOFACIAL SURGERY CLINICS OF NORTH AMERICA

FORTHCOMING ISSUES

September 2013

**Office Procedures for the
Oral and Maxillofacial Surgeon**
Stuart E. Lieblich, DMD, *Editor*

March 2014

Contemporary Rhytidectomy
Landon D. McLain, MD, DMD, *Editor*

PREVIOUS ISSUES

September 2012

Contemporary Management of Third Molars
Louis K. Rafetto, DDS, *Editor*

March 2012

**Digital Technologies in Oral and Maxillofacial
Surgery**
Gary Orentlicher, DMD, *Editor*

September 2011

**Current Concepts in Temporomandibular Joint
Surgery**
Gregory M. Ness, DDS, *Editor*

RELATED ISSUES

Oral and Maxillofacial Clinics of North America November 2012 (Vol. 24, No. 4)
The Orbit
Stephen A. Schendel, MD, DDS, *Editor*

THE CLINICS ARE NOW AVAILABLE ONLINE!

Access your subscription at:
www.theclinics.com

Preface

Craniomaxillofacial Trauma



David A. Bitonti, DMD, CAPT, DC, USN
Editor

The management of maxillofacial trauma and the practice of oral and maxillofacial surgery are integrally linked. The synergistic combination of an intricate understanding of occlusion, functionality of the masticatory system, manual dexterity, and familiarity with surgery in the maxillofacial complex are the strength of the oral and maxillofacial surgeon's treatment, care, and contribution to the management of maxillofacial trauma. Additionally, the unique practice and management of maxillofacial trauma is a combination of sound, tested, surgical principles combined with surgeon ingenuity, flexibility, and adaptability to each individual trauma patient and the injuries with which they present. Due to the nature of maxillofacial trauma, each trauma patient is distinctive, because each of their injuries is distinctive, even when some commonality may exist in injury patterns.

This issue of *Atlas of the Oral and Maxillofacial Surgery Clinics of North America* is intended to provide a review of maxillofacial trauma covering the concepts of sound, tested, surgical principles with the addition of surgeon ingenuity. Included in the articles are lessons learned and anecdotes based on the clinical and surgical experience of the authors and that convey surgeon ingenuity. As the role of the oral and maxillofacial surgeon increases in response to national and

international humanitarian and disaster efforts, it was important to include information pertinent to the delivery of maxillofacial trauma care in that modified or austere environment. It is often so different from what the oral and maxillofacial surgeon's normal trauma care experience is that it warranted a separate article to stimulate thoughtful consideration when planning for and entering into that treatment evolution. Additionally, it was important to cover all the areas of the maxillofacial complex that might be encountered and require evaluation and recognition by the oral and maxillofacial surgeon. In that regard, incorporation of the expertise provided by our colleagues in radiology and oral radiology; maxillofacial prosthetics; ophthalmology; neurosurgery; general surgery; and otolaryngology, head and neck surgery was considered an important component of this edition.

The articles are organized to include general trauma management, diagnostic aids, aspects of injury related to other than blunt trauma, factors related to optimum management including wound care and nutrition, progressive management of injuries from the bottom up, inside to out, and finally, reconstruction. I am pleased and honored by the opportunity to work with the article authors. Each of them brings their own individual experience to the topics. As one delivers maxillofacial trauma care, it is my hope and belief

that the reader will find this edition valuable personally and professionally.

In closing, I want to thank and extend my deepest appreciation to each of the authors for their hard work and diligence in the preparation of this edition. I believe they have done an admirable job in preparing an informative edition for the maxillofacial trauma provider. I want to thank my colleagues who continue to influence me personally and professionally. Most importantly, I thank my family for their constant love and support, especially my wife, Lisa; son, David Joseph; and daughter, Alexandra. They are my inspiration to be a better person, surgeon, husband, and father.

David A. Bitonti, DMD, CAPT, DC, USN
Walter Reed National Military Medical Center
8901 Wisconsin Avenue
Bethesda, MD 20889-5600, USA

E-mail address:
David.A.Bitonti@gmail.com

The views expressed are those of the author and not necessarily those of the Department of Defense, United States Navy, United States Navy Bureau of Medicine and Surgery, or the United States Navy Dental Corps.

Initial Management of the Trauma Patient

J. Michael Ray, DDS^{a,*}, Ramon F. Cestero, MD^b

KEYWORDS

• Trauma • Initial management • Advanced trauma life support

KEY POINTS

- Trauma is the leading cause of death for individuals in the United States up to the age of 45, and is the third leading cause of death overall for all ages.
- The most widely accepted standard of care for initial assessment and treatment of injured casualties is the Advanced Trauma Life Support (ATLS) program.
- Regardless of the injuries sustained or the capabilities of the treating facility, the principles described in ATLS should guide the initial assessment, resuscitation, and treatment of the multiply injured patient.
- The primary and secondary survey should be continually repeated to identify deterioration in the patient's condition and to make appropriate interventions.
- The use of a prioritized and systematic approach to initial management of the trauma patient ensures that optimal care is delivered and the best possible outcome is achieved.

Introduction

Trauma is the leading cause of death for individuals in the United States up to the age of 45, and is the third leading cause of death overall for all ages.¹ Worldwide, trauma is responsible for more than 3 million deaths and 300 million injuries annually,² making it a significant, yet preventable global public health issue.

The most widely accepted standard of care for initial assessment and treatment of injured casualties is the Advanced Trauma Life Support (ATLS) program,³ developed by the American College of Surgeons. It places priority on diagnosis and management of the injuries that are the greatest threat to life first, using a simple ABCDE mnemonic as follows: Airway with C-spine protection, Breathing and ventilation, Circulation with hemorrhage control, Disability—neurologic status, and Exposure and environmental control.

This article focuses on the initial evaluation of the trauma patient, incorporating many of the recent significant changes in management, and addresses the common injuries that may be evaluated by the oral and maxillofacial surgeon.

Airway with C-spine protection

Airway assessment

Verification of a patent airway is paramount during initial evaluation of the trauma patient, because all other

resuscitative efforts are futile without adequate oxygenation and ventilation. All patients should receive high-flow oxygen on initial arrival, and the cervical spine should be immobilized by use of a hard collar or in austere settings, using sand bags secured with tape.

Assessment of the airway typically begins by encouraging the patient to speak, commonly performed by asking "What is your name?" Not only does this allow the physician to evaluate the airway status, but in addition it provides a rapid assessment of mentation if the patient answers in a logical manner. Signs of airway obstruction including stridor, gurgling, agitation, and hoarseness should be quickly assessed. In addition, the physician should evaluate for possible facial, mandibular, or tracheal or laryngeal fractures, which may compromise the airway and eventually lead to obstruction. The presence of blood, vomit, fractured teeth, or other debris in the oral cavity is concerning for potential airway compromise and should be monitored closely.

Neck trauma

All patients with penetrating injury to the neck should be assessed for airway compromise, because potential vascular injury can lead to significant hemorrhage resulting in airway displacement and obstruction. Signs of direct airway injury can include shortness of breath and hemoptysis, and a large neck hematoma with tracheal deviation should prompt urgent intubation before loss of airway.

Maxillofacial trauma

Trauma to the maxillofacial region can cause airway compromise because of hemorrhage, tissue swelling, and fractures leading to loss of facial architecture. Midface injuries can compromise the nasopharynx and oropharynx as a result of fractures and dislocations. Severely comminuted or bilateral mandibular fractures may cause airway obstruction because of collapse of the glottic structures on the posterior pharynx. Dentoalveolar fractures, in addition to being associated with hemorrhage, can be problematic if teeth are dislodged

Disclaimer: The views expressed are those of the author and not necessarily those of the Department of Defense, United States Navy, United States Navy Bureau of Medicine and Surgery, or the United States Navy Dental Corps.

^a Baylor College of Dentistry, Department of Oral and Maxillofacial Surgery, 3302 Gaston Avenue, Dallas, TX 75246, USA

^b Naval Medical Research Unit, Medical Corps, United States Navy, 3650 Chambers Pass, Fort Sam Houston, San Antonio, TX 78234-6315, USA

* Corresponding author.

E-mail address: mray@bcd.tamhsc.edu

because these can be easily aspirated. Therefore, all teeth should be accounted for to ensure none have been aspirated.

Airway management

Indications for intubation of the trauma patient include airway obstruction; shock; altered mental status (Glasgow Coma Scale [GCS] ≤ 8); and occasionally combativeness requiring sedation for evaluation. Initial management of the patient with airway compromise who requires intubation consists of a chin-lift or jaw-thrust maneuver, which is maintained until intubation is achieved. Oropharyngeal airways can serve as helpful adjuncts, but these cannot be used in conscious patients because of potential gagging, vomiting, and aspiration. Nasopharyngeal airways are more tolerable in the awake patient and may transiently aid in maintaining airway patency.

When a decision has been made to initiate a definitive airway, orotracheal intubation is typically performed, although this can be difficult in the setting of bleeding or vomiting because of lack of clear visualization of the cords. It is imperative that all intubation equipment is readily available during initial examination because the need for an emergency airway can develop quickly during initial evaluation. In the urgent setting where orotracheal intubation is unsuccessful, prompt transition to a surgical airway (cricothyroidotomy) is recommended.

Breathing and ventilation

Assessment of breathing and ventilation includes inspection, palpation, and auscultation of the neck, thoracic region, and upper abdomen and back. Injuries that can be identified during the primary survey and may restrict adequate ventilation include tension pneumothorax; flail chest (three or more consecutive ribs fractured in two places) with underlying pulmonary contusions; open pneumothorax; and massive hemothorax. Inspection identifies contusions, penetrating injuries, open wounds and soft tissue defects, flail segments, and asymmetry in chest expansion during inspiration. Palpation may elicit areas of tenderness, subcutaneous emphysema, abnormal chest wall motion, and bony abnormalities. Auscultation, although sometimes difficult in a noisy resuscitation area, can confirm the presence of bilateral breath sounds and when abnormal, can suggest the possibility of a pneumothorax or hemothorax.

Perhaps the most critical abnormality to recognize during this phase is a tension pneumothorax, a true emergency that clinically presents with unilateral absence of breath sounds, tracheal deviation, distended neck veins, and hypotension. A tension pneumothorax develops when air enters the pleural space from the trachea, bronchi, lungs, or chest wall. The air cannot escape, and the progressive increase in pressure in the affected side collapses the lung and mediastinal structures to the contralateral side. As air accumulates and the intrathoracic pressure increases, the mediastinal structures including the heart, superior vena cava, and inferior vena cava are compressed; venous return decreases; and hypotension ensues. Treatment of a tension pneumothorax is relatively simple, consisting of needle decompression above the rib in the second intercostal space along the midclavicular line. This relieves the increased pressure in the pleural cavity, and is confirmed by a rush of air on needle insertion. A thoracostomy tube is then placed to manage the resulting simple pneumothorax, and to prevent reaccumulation of air leading to another tension pneumothorax.

A simple pneumothorax is caused by the entry of air into the thoracic cavity from the chest wall, lung, or trachea, which removes the normal negative pleural pressure maintaining lung expansion and causes the lung to collapse. Clinical findings include decreased breath sounds on the affected side, but physical findings may be subtle if the pneumothorax is small. Pneumothoraces with minimal findings are usually identified on subsequent chest radiograph, and treatment consists of tube thoracostomy.

Similarly, a hemothorax (blood accumulated in the pleural cavity) presents with decreased breath sounds on the affected side because the contained blood prevents full expansion of the lung during inspiration. In the setting of significant bleeding over 1500 mL, patients can present with hypotension in addition to abnormal breath sounds, and this is defined as a massive hemothorax. In patients presenting with stable vital signs, diagnosis is usually made during radiologic evaluation, including chest radiograph or CT scan. In both situations, treatment is tube thoracostomy.

Circulation with hemorrhage control

Shock

After the airway is secured and ventilation has been assessed, the patient's circulatory status is addressed. Shock, defined as inadequate tissue perfusion, can be categorized into four types: (1) hemorrhagic (or hypovolemic); (2) cardiogenic; (3) septic; and (4) neurogenic. The most common cause of shock in the injured patient is hemorrhagic in nature, although neurogenic shock can also be present in the setting of spinal cord injury, and cardiogenic or septic shock can occasionally be seen. It is of utmost importance to recognize the patient in the shock state, because early recognition and treatment is crucial during the primary survey.

Clinical signs of shock include tachycardia; dyspnea; cool and clammy skin; mental status changes; decreased pulse pressure; and in more severe cases, hypotension. Estimations of overall blood loss using vital signs has been suggested by ATLS to assist in determining optimal resuscitation strategies for patients in shock, and degree of shock has been classified into four classes (Classes 1–4) (Table 1). As the severity of shock increases, recommendations for fluid replacement change from crystalloids to packed red blood cells (PRBC) and fresh frozen plasma (FFP).

Identification and control of bleeding source

The circulation and hemorrhage control phase of the primary survey centers around identification of the source of blood loss, controlling ongoing hemorrhage, and replacing the volume loss (Fig. 1). Two large-bore intravenous (IV) lines are initially placed, and bleeding from external wounds is typically controlled with direct pressure. Tourniquets, used much more frequently in military settings, are an excellent adjunct to control severe bleeding from extremities, and have been shown to be extremely effective.⁴

In addition to obvious bleeding from external sources, other sources of bleeding need to be considered during initial evaluation of the patient in shock. These include bleeding from the thorax (massive hemothorax, vascular injury, penetrating cardiac injury); abdomen (solid organ injury [liver, spleen, or kidney], major vessel injury, or mesenteric bleeding);

Table 1 Classes of shock

Class	Blood Loss	Findings	Fluid Replacement
I	<15% (<750 mL)	P < 100, normal BP, normal PP	Crystalloid
II	15%–30% (750–1500 mL)	P = 100–120, normal BP, decreased PP	Crystalloid
III	30%–40% (1500–2000 mL)	P = 120–140, decreased BP, decreased PP	Crystalloid and blood
IV	>40% (>2000 mL)	P > 140, decreased BP, decreased PP	Crystalloid and blood

As the amount of blood loss increases, vital sign abnormalities become more evident. Note that even in class II shock with up to 30% blood loss the blood pressure can be normal and only the pulse and pulse pressure are abnormal. Only in class III shock with up to 2000 mL of blood loss is the blood pressure clearly abnormal.

Abbreviations: BP, blood pressure; P, pulse; PP, pulse pressure.

retroperitoneum (pelvic fracture); or long bone fractures (eg, femur). Chest radiograph is a readily obtainable diagnostic study that provides significant information regarding thoracic sources of shock, because a large hemothorax can be easily recognized on a plain film. A focused abdominal sonographic examination for trauma (FAST) is a sensitive procedure used to determine the presence of fluid in the abdominal cavity, which is typically assumed to be blood until proved otherwise. In a patient with hypotension and a positive FAST, a laparotomy is indicated to identify and control the source of abdominal bleeding. A pelvic plain film radiograph can identify a pelvic fracture with possible retroperitoneal hemorrhage, and either physical examination findings or extremity radiographs can detect the presence of long bone fractures.

Volume replacement

Volume replacement is initiated after securing IV access, and typically consists of a warmed 1- to 2-L bolus of lactated Ringer solution or normal saline. Depending on the patient's response, further resuscitation fluids may consist of more crystalloid, PRBC, or FFP. The amount of fluid or blood products required is difficult to determine on initial evaluation, and therefore the



Fig. 1 Resuscitation of a patient with a penetrating chest injury. The patient is receiving supplemental oxygen by facemask and undergoing a FAST examination and tube thoracostomy.

response to fluid challenges is a major determinant of further infusion of crystalloid or blood products. Parameters that are important to observe after administration of resuscitation fluids include improvement of tachycardia, normalization of blood pressure, clearing of mental status, improved urine output, and overall evidence of improved end-organ perfusion. If the patient only experiences a minimal response or a transient response to fluid administration, this is evidence of ongoing bleeding and further resuscitative strategies should consist of blood products instead of crystalloid fluids.

When a decision has been made to provide blood products, O-positive blood for men or O-negative blood for women is usually readily available for immediate infusion until type-specific, crossmatched blood is obtained from the blood bank. Based largely on military experience during Operation Iraqi Freedom,⁵ PRBC, plasma, and platelets are now provided in a 1:1:1 manner (6 U PRBC:6 U FFP:6-pack platelets or 1 U apheresis platelets) to better replace the components that are being lost during hemorrhage.

Hemostatic resuscitation and permissive hypotension

Hemostatic resuscitation refers to the use of restrictive fluid therapy to maintain a blood pressure that provides adequate end-organ perfusion, but does not increase the blood pressure excessively to dislodge blood clots and cause further unnecessary bleeding before surgical control. General guidelines recommend a goal systolic blood pressure of 80 to 100 mm Hg, or enough to maintain a palpable radial pulse. This permissive hypotension avoids the use of aggressive high-volume fluid replacement to obtain normal vital signs until surgical control can be obtained. After the bleeding source is identified and controlled, normal blood pressures are then established. Hemostatic resuscitation and permissive hypotension are major aspects of the concept known as damage control resuscitation (DCR), which is described later in this article.

Tranexamic acid

Tranexamic acid (TXA), a synthetic derivative of the amino acid lysine, is an antifibrinolytic agent commonly used in cardiac surgery. Largely based on the results of a recent large, prospective, randomized study evaluating the use of TXA in trauma patients,⁶ TXA has been advocated as an important intervention that can significantly reduce the risk of death in bleeding patients. In the original CRASH-2 study, mortality was markedly improved in trauma patients with the use of TXA (14.5% vs 16%). However, in a subsequent analysis of the CRASH-2 data,⁷ the mortality benefit was only present if TXA was administered less than 3 hours after injury, and mortality actually increased if TXA was given after 3 hours (4.4% vs 3.1%). Clinical studies are currently underway to further delineate the benefit of TXA, although many major trauma centers are currently using TXA early after initial injury.

Disability (neurologic status)

The neurologic component of the primary survey quickly assesses the patient's level of consciousness, pupillary size and reaction, and spinal cord injury level. The level of consciousness is determined using the GCS, which is composed of three criteria: (1) eye, (2) verbal, and (3) motor assessments (Table 2). A GCS score less than eight suggests a potential for inability to

Table 2 Glasgow coma scale

Eye opening response	Spontaneous—open with blinking at baseline	4 points
	Opens to verbal command, speech, or shout	3 points
	Opens to pain, not applied to face	2 points
	None	1 point
Verbal response	Oriented	5 points
	Confused conversation, but able to answer questions	4 points
	Inappropriate responses, words discernible	3 points
	Incomprehensible speech	2 points
Motor response	None	1 point
	Obeys commands for movement	6 points
	Purposeful movement to painful stimulus	5 points
	Withdraws from pain	4 points
	Abnormal (spastic) flexion, decorticate posture	3 points
	Extensor (rigid) response, decerebrate posture	2 points
	None	1 point

This scale is used to rapidly assess a patient's neurologic deficit. Continual reassessment is necessary to monitor any changes.

Data from Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet 1974;2:81–4.

protect the airway, and usually mandates an advanced airway to be placed. Lack of motor movement consistent with a spinal cord injury above the level of the fourth thoracic vertebra places the patient at risk for neurogenic shock, and this can complicate patient assessment in the setting of hypotension if the patient is also experiencing hemorrhagic shock. Further assessment of neurologic defects that are not associated with life-threatening injuries, such as cranial nerve deficits, should be deferred until the secondary survey.

Exposure and environmental control

Patients should be completely exposed during the primary survey to fully examine and identify all injuries. However, prolonged exposure places the patient at risk for hypothermia, and therefore the examination should be completed as quickly as possible and the patient covered with warm blankets. In addition, all IV fluids should be infused through a fluid warmer to minimize iatrogenic causes of hypothermia from cold fluid administration.

Adjuncts to primary survey

Various standard investigations and procedures, such as 12-lead electrocardiography, pulse oximetry, arterial blood gases, hemoglobin and hematocrit, coagulation studies, chest and pelvis radiographs, and gastric and urinary catheterization, may provide additional diagnostic information. If needed, plain films of the chest, abdomen, and pelvis can be performed at this time.

Secondary survey

After the primary survey is completed, major life-threatening injuries have been addressed, and vital signs have been stabilized, the provider begins the secondary survey, which includes a history and complete head-to-toe examination.

A history including the mechanism of injury is important to obtain, because certain types of trauma (falls, motor vehicle accidents, auto vs pedestrian injuries, gunshot wounds, and so forth) have commonly associated patterns of injury, which can alert the provider to particular potential injuries. Current medications are important, especially in the elderly, because anticoagulants can be a major cause of uncontrolled bleeding and β -blockers can reduce cardiac output and mask tachycardia. In a patient who is unable to respond, family members, paramedics, or other injured patients may be a good source of information. A useful mnemonic for obtaining a rapid history is "AMPLE," which examines the following important areas: Allergies, Medications, Past illnesses/pregnancy, Last meal, and Events/environment related to the injury.

The physical examination closely examines every region of the body to accurately identify all wounds and limit the possibility of missed injuries. Beginning with the head and skull, the systematic examination progresses to maxillofacial structures, neck and c-spine, chest, abdomen, perineal region, pelvis and extremities, and neurologic system. Although many of these systems overlap, a systematic and thoughtful approach should be taken to ensure completeness.

Head and skull

The scalp and skull should be examined for lacerations, depressions, contusions, and fractures. The GCS should be reevaluated for any changes that might reflect an evolving brain injury and a resultant increase in intracranial pressure from an intracranial hemorrhage or intracranial hypotension. The patient should be monitored closely for signs of hypoxia caused by increasing intracranial pressure including mental status changes and Cushing triad (hypertension, bradycardia, and irregular breathing). A CT scan of the head is mandatory in the setting of abnormal mental status, low GCS, skull injury, and signs of increased intracranial pressure.

A cranial nerve examination at this point may be appropriate along with examination of the eyes and ears. Although edema from facial injuries may preclude a complete eye examination, the surgeon should be as complete as possible with his or her examination. Abnormal findings during the eye examination may suggest a cranial nerve lesion or a more serious injury, such as a brain or brainstem injury. Gross hearing should be checked at this point.

Maxillofacial region

Maxillofacial injuries are not typically life-threatening, so evaluation of injuries that do not involve airway obstruction or significant bleeding is delayed until the secondary survey. The facial skeleton is palpated for any steps or abnormalities. The ears and nose are examined for otorrhea and rhinorrhea, which is an indication of a basilar skull fracture. A complete intraoral examination should be performed at this point to assess for any lacerations, floor of mouth hematomas, or malocclusion, which may be indicators of alveolar, maxillary, or mandibular fractures. A maxillofacial CT scan is useful in evaluating facial

fractures when facial edema precludes a complete and accurate clinical examination.

Neck and cervical spine

Patients who are victims of blunt mechanisms must also undergo cervical spine injury evaluation and stabilization with appropriate immobilization devices, such as a cervical spine collar. Movement of the cervical spine should be limited, especially during assessment and management of the patient's airway, because this can cause or aggravate a neurologic injury in the setting of cervical spine instability. Plain film radiography and CT scan of the cervical spine identify fractures and dislocations, but may miss ligamentous injuries.

Chest

The chest should be reassessed during the secondary survey for non-life-threatening injuries. The chest wall should be inspected and palpated for blunt or penetrating injuries. Specifically, the patient should be examined for signs and symptoms of diaphragmatic hernia; myocardial or pulmonary contusions; and tracheobronchial, aortic, or esophageal disruption. Many serious chest injuries, such as adult respiratory distress syndrome and pulmonary contusions, do not become symptomatic until 48 to 72 hours after the initial injury. Therefore, the patient must be continually monitored for the development of respiratory distress after admission.³

Abdomen

The abdomen is examined for signs of penetrating and blunt trauma. If penetrating wounds are identified, they are best managed in the operating room with a laparotomy. Rebound tenderness and abdominal rigidity may be an indicator of blood in the abdomen, which may also warrant a laparotomy. If the patient exhibits abdominal tenderness, although a laparotomy is not indicated, CT of the abdomen allows visualization of the abdominal contents and the retroperitoneum. As discussed in the primary survey, the FAST examination provides rapid assessment of areas of the abdomen where blood is likely to accumulate.

Perineum

The external genitalia in men and women should be examined for lacerations, contusions, and bleeding. Blood at the urethral meatus is a sensitive indicator of urethral injury and must be closely examined before urinary catheter placement. A rectal examination is necessary on all multisystem trauma patients. The examiner is assessing muscular tone, the presence of blood, rectal integrity, and the position of the prostate. Loss of rectal tone indicates a spinal cord injury and should be investigated further. A bimanual examination is necessary in females with lower abdominal pain or signs of injury.

Pelvis and extremities

The pelvis is clinically examined for fracture and instability, which can lead to life-threatening hemorrhage. Blood loss from a pelvic fracture can occur rapidly, so the diagnosis must be made quickly. A plain radiograph of the pelvis aids in diagnosis and guides initial treatment.

The extremities are examined for deformities, swelling, lacerations, contusions, and equal pulses. Plain radiographs are obtained of all extremities with signs of injury to rule out fractures or dislocations. Initial treatment should consist of prompt splinting of fractures and immobilization of injured joints.

Patients who have sustained pelvic or long bone fractures should be monitored after admission for progressive respiratory failure from fat embolism. Fat embolism can occur from any long bone fracture but is more likely to occur in fractures of the femur or pelvis. Morbidity from fat embolism and the development of adult respiratory distress syndrome can be mitigated by early open reduction and internal fixation, thus allowing early mobilization of the patient.

Neurologic system

The trauma patient's neurologic status should be continually monitored during the secondary survey. The GCS provides a rapid and simple means of assessing a patient's cerebral cortex function by examining pupillary activity, verbal understanding and response, and motor coordination. This examination can be compromised by drugs, alcoholic intoxication, or sedative medications, so continued reassessment is necessary. If not performed during examination of the head and face, a complete cranial nerve examination should be performed. Specifically, the eyes should be closely examined for light reactivity, extraocular movements, and visual acuity.

The patient should remain on a long spine board wearing a rigid cervical collar until spine injury has been ruled out by clinical and radiographic examination. This is especially important when moving or rolling the patient. However, the spine board should be removed as soon as possible to prevent pressure sores. The entire spine is palpated to assess for any deformities, swelling, tenderness, or any penetrating wounds. In a conscious patient, motor function is assessed. If a spinal cord injury has occurred, motor and sensory evaluations are carefully performed to determine the level of paraplegia or quadriplegia.

The extremities are checked for muscular tone and strength, reflexes, and sensation. These must be continually reassessed, and any changes must be documented.

Adjuncts to secondary survey

In the stable patient who does not require immediate surgical intervention, further diagnostic studies follow the completion of the secondary survey. CT scans of the chest, abdomen, and pelvis with IV contrast not only identify significant injuries, such as pneumothorax, hemothorax, pneumoperitoneum, solid organ injury, and pelvic fractures, but also provide information on possible vascular injuries, such as aortic disruption and pelvic arterial bleeding. Interventional radiologic techniques have essentially converted the management of pelvic arterial bleeding from an open procedure with significant blood loss, to a percutaneous intervention, which directly identifies and embolizes the bleeding arterial source. Although each modality is useful in certain situations, not all patients require all of these studies, and therefore these investigations and procedures should be tailored to the individual patient situation.

Damage control resuscitation

DCR is a recently developed concept that encompasses several aspects of trauma resuscitation and management under one overall treatment paradigm. The overall concept can be subdivided into five individual areas: (1) permissive hypotension, (2) prevention and treatment of hypothermia, (3) treatment of acidosis, (4) balanced blood product resuscitation, and (5) damage control surgery (DCS).

Permissive hypotension

Permissive hypotension, or "hypotensive resuscitation," is the goal of maintaining the blood pressure during resuscitation low enough to allow end organ perfusion, but not so high that exsanguination is aggravated before surgical intervention and control of surgical bleeding. In this manner relative hemostasis can be maintained and the patient will not "pop the clot" on the injured vessels because of higher pressures before surgical control or repair.

Unfortunately, no evidence-based recommendations exist from any of the major trauma organizations regarding specific blood pressure ranges to use in permissive hypotension protocols. Data suggest that maintaining systolic blood pressure close to 90 mm Hg prevents rebleeding from recently clotted vessels,^{8–12} but pressures below 80 mm Hg may be inadequate in the setting of head injury.^{13,14} Currently, military field casualty care recommendations suggest the maintenance of mentation or a palpable peripheral pulse as a substitute for blood pressure in the field until a blood pressure can be accurately obtained.¹⁵ After blood pressure can be obtained, current recommendations are to resuscitate to a systolic blood pressure of 90 mm Hg or a mean arterial pressure of 60 mm Hg until definitive surgical control of bleeding is achieved.¹⁶

Prevention and treatment of hypothermia

Hypothermia is an independent risk factor for mortality, and severe hypothermia ($<32^{\circ}\text{C}$) has been associated with a mortality rate approaching 100%.^{17,18} Various causes are responsible for hypothermia in the trauma patient, including bleeding and exposure to a cold environment, altered central thermoregulation, decreased shivering, and decreased heat production.^{19,20} Perhaps the most preventable cause is administration of cold resuscitation fluids,²¹ and therefore warmed fluids are always recommended during resuscitation.

DCR emphasizes not only the aggressive correction of hypothermia but also its prevention, and both goals can be achieved by a combination of passive and active warming methods. Passive warming methods consist of simple techniques, such as removing wet clothing, moving the patient to a warm environment, and providing insulation during resuscitation. Active warming methods include the use of heated blankets, warmed IV fluids, forced-air warming systems, and the maintenance of a heated resuscitation room and operating suite.

Treatment of acidosis

Severe acidosis is associated with multiple detrimental physiologic abnormalities including bradycardia, hypotension, decreased contractility, reduced cardiac output, and abnormalities in coagulation.^{22–24} Metabolic acidosis is a significant

byproduct of hemorrhagic shock,²⁵ and multiple studies have shown an association between acidosis with coagulopathy and poor outcomes in trauma patients.^{26–29}

Because the metabolic acidosis in trauma is caused by significant blood loss and hypoperfusion of end organs, correction of acidosis requires eventual restoration of intravascular volume after control of hemorrhage. In DCR, restoration of end-organ perfusion is ultimately accomplished by balanced 1:1:1 blood product resuscitation (discussed next). However, this can only be satisfactorily achieved after hemorrhage has been controlled, so other treatments to correct acidosis are instituted until hemostasis is established.

Balanced blood product resuscitation (1:1:1)

To rapidly and effectively correct the significant physiologic alterations experienced by the severely injured trauma patient, fluid resuscitation in DCR consists of using PRBCs, plasma (FFP or thawed plasma), and platelets in a balanced 1:1:1 manner. Military studies conducted during the Iraq war suggested that patients receiving higher ratios of FFP to PRBCs (1:1.4) had lower mortality rates (19% vs 65%) compared with those who received lower ratios (1:8).⁵ Similar results have been found in the civilian sector, where a retrospective analysis of patients receiving massive transfusions found a lower mortality (26% vs 87.5%) in those who received FFP:PRBC in a higher ratio (1:1 vs 1:4).³⁰

Damage control surgery

The concept of DCS was developed as a result of the poor outcomes noted during traditional approaches to severe traumatic hemorrhage, where the triad of hypothermia, acidosis, and coagulopathy led to death during attempts to perform prolonged definitive surgery.

DCS is currently defined in the US Emergency War Surgery textbook as "the rapid initial control of hemorrhage and contamination, temporary closure, resuscitation to normal physiology in the ICU, and subsequent re-exploration and definitive repair."¹⁶ After patients are resuscitated in the emergency department and transported to the operating room, an abbreviated operation is performed with the goals of initially controlling surgical hemorrhage and then limiting contamination from gastrointestinal sources. The patient then undergoes a temporary abdominal closure; is brought to the intensive care unit; and the conditions of hypothermia, acidosis, and coagulopathy are corrected by the application of DCR principles. When normal physiologic parameters are achieved, typically within 24 to 36 hours after operation, the patient is returned to the operating room for definitive repair of all injuries.

Summary

Regardless of the injuries sustained or the capabilities of the treating facility, the principles described in ATLS should guide the initial assessment, resuscitation, and treatment of the multiply injured patient.³ The primary and secondary survey should be continually repeated to identify deterioration in the patient's condition and to make appropriate interventions. The use of a prioritized and systematic approach to initial management of the trauma patient ensures that optimal care is delivered and the best possible outcome is achieved.

References

1. Web-based Injury Statistics Query and Reporting System (WISQARS) [online]. In: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control <http://cdc.gov/injury/wisqars/fatal.html>.
2. Murray C, Lopez A. Global health statistics, vol. 2. Cambridge (MA): Harvard School of Public Health; 1996.
3. Advanced trauma life support. 8th edition. Chicago (IL): American College of Surgeons; 2008.
4. Beekley AC, Sebesta JA, Blackburne LH, et al. Prehospital tourniquet use in Operation Iraqi Freedom: effect on hemorrhage control and outcomes. *J Trauma* 2008;64(Suppl 2):S28–37 [discussion: S37].
5. Borgman MA, Spinella PC, Perkins JG, et al. The ratio of blood products transfused affects mortality in patients receiving massive transfusions at a combat support hospital. *J Trauma* 2007;63(4):805–13.
6. Shakur H, Roberts I, Bautista R, et al. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial. *Lancet* 2010;376(9734):23–32.
7. Roberts I, Shakur H, Afolabi A, et al. The importance of early treatment with tranexamic acid in bleeding trauma patients: an exploratory analysis of the CRASH-2 randomised controlled trial. *Lancet* 2011;377(9771):1096–101. 1101.e1–2.
8. Rhee P, Koustova E, Alam HB. Searching for the optimal resuscitation method: recommendations for the initial fluid resuscitation of combat casualties. *J Trauma* 2003;54(Suppl 5):S52–62.
9. Bickell WH, Wall Jr MJ, Pepe PE, et al. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. *N Engl J Med* 1994;331(17):1105–9.
10. Burris D, Rhee P, Kaufmann C, et al. Controlled resuscitation for uncontrolled hemorrhagic shock. *J Trauma* 1999;46(2):216–23.
11. Dutton RP, Mackenzie CF, Scalea TM. Hypotensive resuscitation during active hemorrhage: impact on in-hospital mortality. *J Trauma* 2002;52(6):1141–6.
12. Sondeen JL, Coppes VG, Holcomb JB. Blood pressure at which rebleeding occurs after resuscitation in swine with aortic injury. *J Trauma* 2003;54(Suppl 5):S110–7.
13. Greaves I, Porter KM, Revell MP. Fluid resuscitation in pre-hospital trauma care: a consensus view. *J R Coll Surg Edinb* 2002;47(2):451–7.
14. Henry S, Scalea TM. Resuscitation in the new millennium. *Surg Clin North Am* 1999;79(6):1259–67. viii.
15. Tactical Combat Casualty Care Guidelines, Committee on Tactical Combat Casualty Care, Military Health System, September 2012.
16. Borden Institute. Emergency war surgery. 3rd U.S. revision edition. Washington, DC: Office of the Surgeon General, U.S. Army, Borden Institute, Walter Reed Army Medical Center; 2004.
17. Jurkovich GJ, Greiser WB, Luteran A, et al. Hypothermia in trauma victims: an ominous predictor of survival. *J Trauma* 1987;27(9):1019–24.
18. Morris Jr JA, Eddy VA, Blinman TA, et al. The staged celiotomy for trauma. Issues in unpacking and reconstruction. *Ann Surg* 1993;217(5):576–84 [discussion: 584–6].
19. Kirkpatrick AW, Chun R, Brown R, et al. Hypothermia and the trauma patient. *Can J Surg* 1999;42(5):333–43.
20. Tsuei BJ, Kearney PA. Hypothermia in the trauma patient. *Injury* 2004;35(1):7–15.
21. Shapiro MB, Jenkins DH, Schwab CW, et al. Damage control: collective review. *J Trauma* 2000;49(5):969–78.
22. Mikhail J. The trauma triad of death: hypothermia, acidosis, and coagulopathy. *AACN Clin Issues* 1999;10(1):85–94.
23. Meng ZH, Wolberg AS, Monroe III DM, et al. The effect of temperature and pH on the activity of factor VIIa: implications for the efficacy of high-dose factor VIIa in hypothermic and acidotic patients. *J Trauma* 2003;55(5):886–91.
24. Ho AM, Karmakar MK, Dion PW. Are we giving enough coagulation factors during major trauma resuscitation? *Am J Surg* 2005;190(3):479–84.
25. Rotondo MF, Zonies DH. The damage control sequence and underlying logic. *Surg Clin North Am* 1997;77(4):761–77.
26. Schreiber MA, Perkins J, Kiraly L, et al. Early predictors of massive transfusion in combat casualties. *J Am Coll Surg* 2007;205(4):541–5.
27. Cosgriff N, Moore EE, Sauaia A, et al. Predicting life-threatening coagulopathy in the massively transfused trauma patient: hypothermia and acidosis revisited. *J Trauma* 1997;42(5):857–61 [discussion: 861–2].
28. Davis JW, Parks SN, Kaups KL, et al. Admission base deficit predicts transfusion requirements and risk of complications. *J Trauma* 1996;41(5):769–74.
29. Davis JW, Kaups KL. Base deficit in the elderly: a marker of severe injury and death. *J Trauma* 1998;45(5):873–7.
30. Duchesne JC, Hunt JP, Wahl G, et al. Review of current blood transfusions strategies in a mature level I trauma center: were we wrong for the last 60 years? *J Trauma* 2008;65(2):272–6 [discussion: 276–8].

The Treatment of Maxillofacial Trauma in Austere Conditions

J. Michael Ray, DDS

KEYWORDS

• Maxillofacial trauma • Oral surgery • Disaster • Triage

KEY POINTS

- When possible, disaster-response teams and equipment should be identified and organized before the incident occurs.
- Triage is a difficult but necessary component of disaster-relief and combat casualty management.
- Even in austerity, sound surgical principles and an attempt to achieve a high standard of care should guide the surgeon.
- Caring for severely injured patients, whether they be in the theater of combat or after a natural disaster, can be a rewarding and even life-changing experience for all involved.

Introduction

Oral and maxillofacial surgeons are routinely called on to provide surgical care for severely injured patients. Occasionally, those times may be under extreme circumstances, such as after a natural disaster or in the theater of war. At times like these, even the most highly skilled and experienced surgeon meets significant challenges trying to care for injured patients. This article describes some of the challenges and unique considerations a surgeon and their team encounter in treating these injuries in austere conditions.

Preparation

The process of establishing disaster-response teams and military forward surgical teams extends beyond the scope of this article and likely beyond the responsibility of the oral and maxillofacial surgeon. However, many critical points should still be considered. When possible, personnel and specific roles should be identified, supplies made accessible, and relationships with governmental and nongovernmental organizations established before the incident occurs.^{1,2}

The surgeon treating facial injuries is usually a part of a larger surgical team that includes multiple surgical and medical specialists. Many surgeons of different specialties selflessly volunteered their services after the Haiti earthquake of 2010, but many were frustrated because finding a place to serve was difficult and often not possible. Although surgical specialists were readily available, nurses and support staff were critically short. In wartime or other military operations, the oral and maxillofacial surgeon is typically located at a well-equipped, higher-echelon facility, either afloat or ashore, with well-trained and adequate numbers of support personnel.

Hospital facilities vary with the circumstances of each disaster. Whether the operating room and wards are located in temporary structures or in existing facilities, strict adherence to universal precautions and aseptic technique is essential. Highly communicable diseases such as typhoid and tuberculosis are endemic in many areas of the world, and isolation is difficult at best for those patients who are suspected of having a contagious disease. The World Health Organization recommends that all health care workers be immunized against hepatitis A, hepatitis B, polio, diphtheria, tetanus, and typhoid, at a minimum.³ Sterilization of surgical instruments is challenging, but many options are available. Instruments must be scrubbed free of organic matter before being sterilized. When possible, autoclaving is the preferred method of sterilizing. If autoclaving is not possible, dry heat or antiseptic (cold sterilization) methods are acceptable.⁴ Some recommendations are listed in [Box 1](#).

Initial management

When multiple severely injured patients require treatment, it is important to properly sort and organize treatment of the casualties, based not only on the severity and survivability of the injuries but also on the capabilities of the facility. Triage is a stressful but necessary part of disaster-relief management. Meaningful rationing of resources, when absolutely necessary, may reduce overall morbidity and mortality and ensure a sense of fairness to all.¹ Ideally, triage is performed by the most experienced trauma surgeon available. If this practice is not possible, the surgeon charged with the responsibility of triage should be trained and experienced in treating patients who have sustained multisystem injuries, regardless of their specialty.

Initial stabilization is performed by anesthesia, surgery, and emergency department personnel. The American College of Surgeons' primary and secondary survey as described in *Advanced Trauma Life Support for Doctors* (ATLS) is an excellent method of initial assessment and resuscitation and is appropriate in both disaster-relief and wartime scenarios. This

Baylor College of Dentistry, Department of Oral and Maxillofacial Surgery, 3302 Gaston Avenue, Dallas, TX 75246, USA
E-mail address: mray@bcd.tamhsc.edu

Box 1. Sterilization techniques

1. Autoclaving
 - a. Autoclaving should be the main form of sterilization, when possible.
 - b. Before sterilizing medical items, they must first be disinfected and vigorously cleaned to remove all organic material. Proper disinfection decreases the risk for the person who cleans the instruments.
 - c. Sterilization of all surgical instruments and supplies is crucial in preventing transmission of human immunodeficiency virus (HIV). All viruses, including HIV, are inactivated by steam sterilization (autoclaving) for 20 minutes at 121°C to 132°C or for 30 minutes if the instruments are in wrapped packs.
 - d. Appropriate indicators must be used each time to show that sterilization has been accomplished. At the end of the procedure, the outsides of the packs of instruments should not have wet spots, which may indicate that sterilization has not occurred.
2. Dry heat
 - a. If items cannot be autoclaved, they can be sterilized by dry heat for 1 to 2 hours at 170°C. Instruments must be clean and free of grease or oil.
 - b. Sterilizing by hot air is a poor alternative to autoclaving, because it is suitable only for metal instruments and a few natural suture materials.
 - c. Boiling instruments is now regarded as an unreliable means of sterilization and is not recommended as a routine in hospital practice.
3. Antiseptics
 - a. In general, instruments are no longer stored in liquid antiseptic. However, sharp instruments, other delicate equipment, and certain catheters and tubes can be sterilized by exposure to formaldehyde, glutaraldehyde, or chlorhexidine.
 - b. If you are using formaldehyde, carefully clean the equipment and then expose it to vapor from paraformaldehyde tablets in a closed container for 48 hours. Ensure that this process is performed correctly.
 - c. Glutaraldehyde is a disinfectant that is extremely effective against bacteria, fungi, and a wide range of viruses. Always follow the manufacturer's instructions for use.

Data from World Health Organization. Best Practice Guidelines on Emergency Surgical Care in Disaster Situations. World Health Organization, 2003.

process is also described in more detail in the article by Ray and Cestero elsewhere in this issue.

Although the treatment of isolated maxillofacial trauma does not usually involve a significant amount of blood loss, transfusion of blood products is often necessary in the severely and multiply injured patient. This is a routine procedure during resuscitations in most hospitals, but it may be a difficult problem in an austere environment. Part of the planning of

a disaster relief or military operation must include provisions for transfusions. If the reliable delivery of blood components or apheresis is difficult, using available staff personnel as a walking blood bank may be considered for the delivery of whole blood for resuscitation.^{5,6}

Once the patient has been stabilized, the secondary evaluation and examination can continue. Because advanced radiologic modalities like computed tomography may not be available, a thorough clinical examination is imperative. However, the surgeon may have conventional plain film capabilities, and these may be useful as an adjunct to a clinical examination. Laboratory analysis of blood or other specimens may also not be available, so the team must rely on a thorough history and physical examination in guiding treatment.

Anesthesia considerations

Administering general anesthesia in an austere environment is a challenge and requires innovation. Although fully equipped anesthesia machines and vaporizers may not be available, several options exist for the anesthesia provider. Total intravenous anesthesia (TIVA) has an advantage over conventional inhalational anesthesia in that less equipment is needed and the medications used may be more readily available. TIVA medications (sedative/hypnotics, ketamine, propofol) are generally safe in the stable patient but may be more difficult to use in the unstable multisystem or combat-injured patient.^{7,8} Smaller, portable volatile anesthetic delivery systems are available and may be suited for both disaster-relief and combat environments.^{8,9}

Surgical considerations

In a disaster-relief or combat environment, achieving a desirable result can be difficult and frustrating. In current and recent theaters of war, the injuries sustained were usually caused by improvised explosive devices (IEDs) and high-velocity gunshot wounds.^{10–12} High-velocity gunshot wounds can cause not only significant disruption of soft and hard tissues but also avulsive wounds (Fig. 1). IEDs can cause not only avulsive wounds but also deep and widespread penetration of dirt, rocks, and metal fragments (Fig. 2). Conversely, injuries caused by the earthquake in Haiti in 2010 were associated with high-energy yet low-velocity crush injuries.¹³ No



Fig. 1 Stellate pattern on injury from a high-velocity gunshot wound.



Fig. 2 Typical appearance of a facial injury caused by an IED blast. Note the penetration of dirt and avulsive nature of the injury.

matter the situation, the general goal of treatment is to return the patient back to preinjury function and appearance and to resume a normal life.

Soft tissue management

Initial treatment of any soft tissue wound should begin with thorough exploration and debridement of any embedded or foreign matter and devitalized tissue. Contaminated wounds should never be primarily closed. Delayed primary closure after multiple debridement and irrigation procedures may be necessary when treating severely contaminated or macerated wounds. Between debridements, wound dressing consisting of saline-moistened gauze placed over the wounds is sufficient to keep the areas covered and allow for the formation of granulation tissue. Moistening the gauze with bleach or antibiotic solutions is not necessary and may delay the formation of granulation tissue. **Box 2** describes the management considerations for the care of contaminated or infected wounds (Figs. 3 and 4).¹⁴

Many wounds cannot be closed primarily, either the amount of wound contamination or because of excessive tissue loss. If a wound is so large that it cannot be closed primarily, the surgeon should seek other options. Smaller wounds should be allowed to completely granulate. The use of negative pressure wound therapy (wound vac) may assist in forming granulation tissue at the tissue bed. This procedure reduces the size of the defect and allows a more suitable recipient site for skin grafting. Local or regional pedicled flaps may be considered as well. Because of the complex instrumentation required, the usual length of operative time, and inability to provide sufficient follow-up, free flaps are generally not recommended.

When dealing with avulsive wounds of the lower face, one of the crucial goals of soft tissue repair is achieving and maintaining intraoral wound closure. Figs. 5–10 show the

Box 2. Soft tissue management guidelines

- Never close infected wounds^a. Systematically perform wound toilet and surgical debridement. Continue the cycle of surgical debridement and saline irrigation until the wound is completely clean.
- Do not close contaminated wounds^b and clean wounds that are more than 6 hours old. Manage these wounds with surgical toilet, leave open, and then close 48 hours later. This procedure is known as delayed primary closure.

To prevent wound infection:

- Restore breathing and blood circulation as soon as possible after injury. Warm the victim, and at the earliest opportunity provide high-energy nutrition and pain relief.
- Perform wound toilet and debridement as soon as possible (within 8 hours if possible).
- Respect universal precautions to avoid transmission of infection.
- Give antibiotic prophylaxis to victims with deep wounds and other indications.
- Antibiotics do not reach the source of the wound infection. Antibiotics reach only the area around the wound; they are necessary but not sufficient and need to be combined with appropriate debridement and wound toilet, as described earlier.
- Use of topical antibiotics and washing wounds with antibiotic solutions are not recommended.

^a An infected wound is a wound with pus present.

^b A contaminated wound is a wound containing foreign or infected material.

Data from World Health Organization. Public health risk assessment and interventions, Earthquake: Haiti, Annex 1. World Health Organization; 2010.



Fig. 3 Nonhealing wound that was not adequately explored and debrided before closure.



Fig. 4 Aggressive exploration revealed multiple fly larvae contaminating the wound.



Fig. 6 Initial presentation of significant soft and hard tissue damage caused by an IED blast.

disappointing sequelae of failed intraoral closure. The inability to cover the mandible and provide a barrier from saliva increases the incidence of infection and further wound breakdown (see Figs. 6–10; Fig. 11). If the surgeon cannot achieve intraoral closure, maxillomandibular or external fixation is recommended until the wounds sufficiently heal by secondary intention to allow for open reduction and internal fixation (ORIF).



Fig. 5 Wound healing well after multiple debridements and delayed primary closure.



Fig. 7 Appearance after debridement and irrigation procedures.



Fig. 8 Open reduction and internal fixation of mandible fracture.



Fig. 9 Further wound breakdown and inability to cover mandibular hardware.



Fig. 10 Raising of pedicled deltopectoral flap to cover lower facial wound.



Fig. 11 Flap inset and wound vac placed over shoulder wound.

Hard tissue management

Not all facial fractures require operative intervention, so the benefits of surgical repair must be weighed against the potential complications as well as the depletion of consumable supplies. Because the resupply of bone plates and screws may be unreliable, ORIF may be reserved for the most debilitating injuries. In a combat environment, ORIF is generally not recommended when evacuation of the patient to a higher level of care is an option. This is typically the case with coalition forces. Initial and intermediate operative management of patients who can be evacuated should consist of securing an airway, control of hemorrhage, maxillomandibular fixation (MMF) or external fixation as needed, and appropriate soft tissue debridement.^{15,16} If evacuation is not possible, the surgeon must decide which method of fracture repair is most appropriate. Immobilization of fractures with MMF is appropriate for many mandibular and maxillary fractures. In patients with a significant amount of comminution or soft tissue loss, MMF or external fixation may be the most appropriate therapy. If sufficient healthy soft tissue is present, ORIF with plates and screws is preferred. Although rarely used, wire osteosynthesis with MMF may be a suitable method of fixation as well. If tissue loss has occurred, the formation of granulation tissue over the bone may occur faster with wire osteosynthesis than over thicker, higher-profile reconstruction plates because of the low profile of the wires (Fig. 12).

Recovery and postoperative management

Wound care, nutrition, and supportive care are at least as important as the initial surgical intervention and must be considered accordingly. Adequate nursing and medical care in the postoperative setting is essential to patient survival. Complex dressings and wound vac changes may be difficult to perform at the bedside and, therefore, may necessitate regular returns to the operating room until wounds can be closed primarily. Reoperation as a result of infection and wound breakdown is an unfortunate event, but this may be necessary and even expected in an austere setting, because of the complexity of the injuries and the difficulty in maintaining adequate aseptic care.

Providing adequate nutrition is essential during the hospitalization and after discharge. Enteral feedings via nasogastric, Dobhoff or gastrostomy tubes may be beneficial initially for severely injured patients, but they may be of minimal use after



Fig. 12 Wire osteosynthesis for a comminuted mandible fracture.

the patient is discharged. Even oral or enteral feedings of puréed food may be impossible because of the lack of electricity and advanced equipment.

Disposition

Disposition and discharge planning begins at the time of admission to the treating facility and is often the most difficult aspect of patient care in the austere environment. The disposition of the patient affects all aspects of the patient's pending care, including initial triage decisions, operative management, medication regimen, nutritional options, physical therapy, and wound care ability. In times of natural disasters, local hospitals or other established medical treatment facilities are often incapable of providing initial care or receiving transfers. If the surgical team chooses to assume the burden of surgical care for severely injured patients, they must be prepared to provide long-term care for these patients until a suitable accepting medical facility can be established. Discharging a patient home is often not an acceptable option because of the confounding circumstances that demanded the team's presence in the first place.

Summary

Caring for severely injured patients, whether they be in the theater of combat or after natural disasters, can be a rewarding and even life-changing experience for all involved. Sound surgical principles and an attempt to achieve a high standard of care should still guide the treating surgeon. The surgical team undoubtedly face numerous obstacles, but with careful and considerate planning, many of these can be minimized.

References

1. Hanfling D, Altevogt BM, Gostin LO. A framework for catastrophic disaster response. *JAMA* 2012;308(7):675–6.
2. Inglesby TV. Progress in disaster planning and preparedness since 2001. *JAMA* 2011;306(12):1372–3.
3. World Health Organization. Public health risk assessment and interventions: earthquake, Haiti. Haiti (Geneva): Communicable Disease Working Group on Emergencies (WHO/HQ) Communicable Disease Surveillance and Response (AMRO/PAHO); WHO Office; 2010.
4. Lichtenberger P, Miskin IN, Dickinson G, et al. Infection control in field hospitals after a natural disaster: lessons learned after the 2010 earthquake in Haiti. *Infect Control Hosp Epidemiol* 2010; 31(9):951–7.
5. Malsby III R, Frizzi J, Ray P, et al. Walking donor transfusion in a far forward environment. *South Med J* 2005;98(8):809–10.
6. Repine TB, Perkins JG, Kauvar DS, et al. The use of fresh whole blood in massive transfusion. *J Trauma* 2006;60(Suppl 6):S59–69.
7. Lewis S, Jagdish S. Total intravenous anaesthesia for war surgery. *J R Army Med Corps* 2010;156(4 Suppl 1):301–7.
8. Mellor J. Anaesthesia in austere environments. *J R Army Med Corps* 2005;151(4):272–6.
9. Gegel BT. A field-expedient Ohmeda Universal Portable Anesthesia Complete draw-over vaporizer setup. *AANA J* 2008;76(3):185–7.
10. Belmont PJ, Schoenfeld AJ, Goodman G. Epidemiology of combat wounds in Operation Iraqi Freedom and Operation Enduring Freedom: orthopaedic burden of disease. *J Surg Orthop Adv* 2010; 19(1):2–7.
11. Belmont Jr PJ, McCrisky BJ, Sieg RN, et al. Combat wounds in Iraq and Afghanistan from 2005 to 2009. *J Trauma Acute Care Surg* 2012;73(1):3–12.
12. Pasquier P, de Rudnicki S, Donat N, et al. Epidemiology of war injuries, about two conflicts: Iraq and Afghanistan. *Ann Fr Anesth Reanim* 2011;30(11):819–27 [in French].
13. Ray JM, Lindsay RW, Kumar AR. Treatment of earthquake-related craniofacial injuries aboard the USNS Comfort during Operation Unified Response. *Plast Reconstr Surg* 2010;126(6):2102–8.
14. World Health Organization. Public health risk assessment and interventions, Earthquake: Haiti, Annex 1. World Health Organization; 2010.
15. Goksel T. Improvised explosive devices and the oral and maxillofacial surgeon. *Oral Maxillofac Surg Clin North Am* 2005;17(3): 281–7. vi.
16. Will MJ, Goksel T, Stone Jr CG, et al. Oral and maxillofacial injuries experienced in support of Operation Iraqi Freedom I and II. *Oral Maxillofac Surg Clin North Am* 2005;17(3):331–9. vii.

Characteristics of Ballistic and Blast Injuries

David B. Powers, DMD, MD, FACS, FRCS (Ed)^{a,*}, Robert I. Delo, DDS, MD^b

KEYWORDS

• Facial • Maxillofacial • Ballistic • Blast • Soft tissue injury • Bone injury

KEY POINTS

- The permanent cavity is the site of initial permanent tissue destruction.
- Deformation of the projectile after impacting hard tissues causes an increase in the size of the permanent cavity.
- After striking bone, fragmentation of the projectile and/or bone can result in the formation of numerous secondary projectiles each producing additional wounding potential, enlarging the size of the permanent cavity.
- The ultimate fate and compositional makeup of the projectile is more important than its velocity or caliber.
- Soft tissue injuries inherent in ballistic trauma may exhibit avulsive loss, sequential necrosis over days to weeks, and compromised vascularity, negating/delaying potential microvascular or pedicled soft tissue reconstruction.

Introduction

Ballistic injury patterns to the craniomaxillofacial region present a unique, and challenging, dilemma for the facial trauma surgeon. The tissue disruption associated with ballistic injury to the head and neck region can be daunting, and the identification of normal anatomic planes, potentially lost within bleeding, destroyed soft and hard tissues can challenge the skills of even the most experienced facial trauma specialist. Although classically considered under the purview of the military trauma surgeon, ballistic and blast injuries also are routinely treated by the civilian surgeon because of the incidence of intentional and unintentional firearm injuries and industrial accidents. A basic understanding of the definitions and characteristic clinical findings of ballistic and blast wounds should be an important tool in the armamentarium of the practicing craniomaxillofacial trauma surgeon.

Introduction to commonly used ballistic terms

Any introduction to the study of ballistic injuries should provide a review of commonly used terms. Table 1 provides the necessary background information to recognize the terminology associated with ballistics, and how those components correlate to an understanding of ballistic injuries:

As historical concepts of ballistics teach that impact kinetic energy is equal to one-half the mass of the projectile times velocity squared ($KE = \frac{1}{2} MV^2$), one would wrongly assume caliber (size) of the projectile and velocity are the sole components of injury calculation. Actually, the increased

energy transmitted from a high-velocity projectile does not necessarily translate to increased wounding capacity, as will be noted throughout the remainder of this article, and the physical properties of the projectile, and its fate on striking the victim, are more important than the caliber. Caliber alone has no alteration to the surgical treatment of the injury, and primarily serves to satisfy the curiosity of the attending medical staff. By understanding the basic mechanical properties of the projectile expelled toward the target, the correlation of velocity and subsequent energy transfer, and the anatomic properties of the head and neck, the craniomaxillofacial trauma surgeon will have a better understanding of the consequences of ballistic injury to the facial skeleton (Box 1).

Yaw, precession, and nutation are frequently referenced when initially studying ballistics. Yaw and precession decrease as the distance of the bullet from the barrel increases, and along with nutation, are terms generally associated with shooting from a distance, as seen in military-grade artillery weaponry (Fig. 1). Illustrative examples of yaw are exaggerated in excess of 30° for graphic representation, while in actuality the degree of yaw is generally less than 1° to 2°, affording tight control of the projectile in flight and allowing the projectile to hit what the firearm is aimed toward. Should the yaw be as exaggerated as seen in most artistic renderings, the projectile would be uncontrollable because of tumbling along the path of fire. A clear clinical example of the effects of yaw and precession is that when examined on a shooting range, projectile holes in targets are consistently circular in nature, indicating the spherical shape of the projectile, not a ragged opening consistent with "tumbling" or excessive yaw. Of interest, projectiles *do* tumble within the body of the target, causing increased damage after striking hard tissue, or with deformation of the projectile. Although these terms have practical applications in weaponry, the clinical significance of these items in craniomaxillofacial ballistic trauma is negligible at best.

The magnus and coriolis effects are also frequently referenced in the discussion of ballistics, but again are isolated to the military applications of projectile flight, as well as the recreational aspects of long-range firing for hunting or competitive shooting. As the overwhelming majority of ballistic

^a Duke Craniomaxillofacial Trauma Program, Division of Plastic, Reconstructive, Maxillofacial and Oral Surgery, Duke University Medical Center, DUMC Box 2955, Durham, NC 27710, USA

^b 59th Medical Wing/Department of Oral and Maxillofacial Surgery, 2200 Bergquist Drive, Suite 1, Lackland AFB, TX 78236, USA

* Corresponding author.

E-mail address: David.Powers@duke.edu

Table 1 Commonly used terms in ballistics

Components of ammunition	
Cartridge/Round	A unit of firearm ammunition
Projectile	The component of the round that is expelled toward the target, sometimes referred to as the "bullet"
Magnum	A cartridge loaded with either a greater volume or more powerful propellant than the original cartridge design, imparting greater velocity to the projectile
Components of weapon	
Rifling	Helical grooves in the barrel of a weapon, which imparts spin along the long axis of the projectile
Caliber	The internal diameter of the barrel of a weapon, usually measured in millimeters or fractions of an inch
Gauge/Bore	The total number of round lead balls that would fill the diameter of the barrel and weigh 1 pound
Associated terms seen in ballistic literature	
Yaw	Movement along the longitudinal access of the projectile
Precession	Rotation of the projectile around the center of mass
Nutation	Small circular movement along the projectile tip
Magnus effect	Lateral crosswind effect of a spinning projectile in flight
Coriolis effect	Spherical shape and rotational properties of the Earth, and its orbit, as it applies to the projectile

injuries to the head and neck region occur within relatively short distances, well within the effective range of the weapon and projectiles, these definitions and concepts have minimal to no correlation to the remainder of this article, or for the surgical management of these ballistic injuries.

Components of ballistic missiles

As previously described, the cartridge or round describes a unit of firearm ammunition. Each round consists of the following (Fig. 2):

- Projectile
- Casing
- Propellant
- Primer

The components of a round provide a basic understanding of the principles of firearm injury. The projectile is the portion of the bullet that is expelled and strikes the target. The compositional makeup of the projectile (soft lead, hollow point, full copper covering, or multiple pellets, as seen in shotguns) has a direct correlation on the wounding potential of the weapon. As a projectile deforms after striking the victim, either as a result of metallurgic composition during manufacturing, or as a direct consequence of striking the underlying bone, the energy transfer to the victim, and potential injury to associated tissues, is increased. As noted earlier, the actual projectiles expelled by firearms are limited in type only by the imagination of the manufacturers and firearm enthusiast. The casing is the

container packaging the projectile, propellant (gunpowder or cordite), and primer as a single unit for placement into the firing mechanism of the weapon. The propellant, such as gunpowder or cordite, is the accelerant that actually allows for expulsion of the projectile from the weapon. The more propellant in a cartridge, as is seen in magnum and rifle rounds, the greater velocity the projectile exhibits. Wadding, or wads, are generally plastic frameworks with a paper or felt insert that hold the various pellets (projectiles) together in relation to the propellant, allowing for accurate and safe release of all the projectiles simultaneously from the barrel in scattershot and shotgun cartridges. Without the presence of wadding, the gas produced by the propellant would push through the pellets, and not propel them as a unit. The primer is the only portion of the bullet with an explosive charge. As the primer is struck by the firing pin of the weapon, the explosive charge is activated, igniting the propellant and sending the projectile on its flight. Some cartridges are referred to as *rimfire*, as the priming mechanism is contained within the rim of the base rather than a separate primer in the center of the base. Generally, rimfire cartridges are less powerful and cannot be reloaded, whereas centerfire cartridges can have the primer replaced and reloaded with another projectile.

Rifles, handguns, and machine guns have rifled barrels; essentially, spiral grooves cut into the length of the interior of the

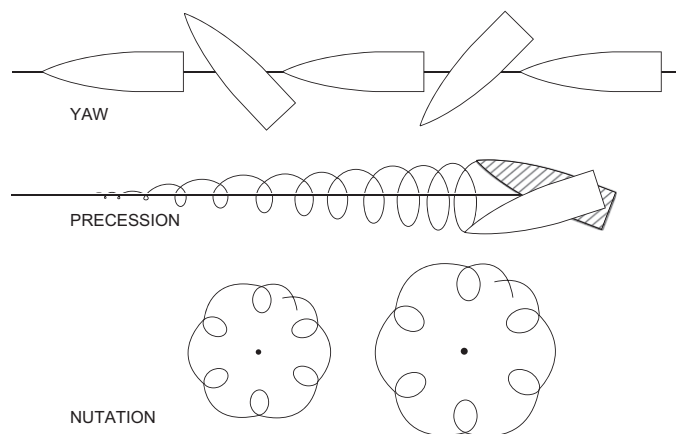


Fig. 1 Yaw: Movement along the longitudinal access of the projectile; precession: rotation of the projectile around the center of mass; nutation: small circular movement along the projectile tip.

Box 1. Factors affecting energy transfer between a projectile and body tissue

Velocity
Profile
Shape
Stability
Fragmentation
Expansion
Secondary impact

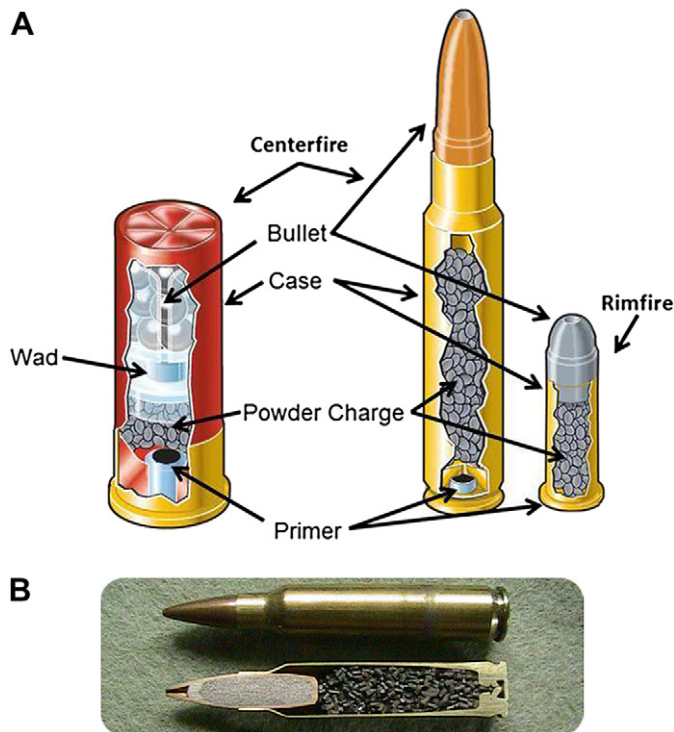


Fig. 2 (A) Cross-sectional analysis of a cartridge and shotgun shell. (B) Cross-section of a full-metal jacketed rifle cartridge.

bore of the barrel. The grooves impart spin upon the projectile, stabilizing it in flight and allowing the projectile to travel in a controlled manner to the target. The grooves are separated by segments of metal, called lands, which project into the middle of the barrel. The diameter of the barrel measured between the lands represents the caliber of the projectile. Caliber specifications based on nomenclature used in the United States can be difficult to comprehend, and utterly confusing to the health care team. The 0.30-06 and the Winchester 0.308 cartridges are both loaded with bullets that have a diameter of 0.308 inches. The "06" in this term describes the year, 1906, when the cartridge was introduced to the market. The term "grains" originally was applied to black powder charges and refers to the weight of the powder in the cartridge, not the number of granules contained in the cartridge case. A 0.30-30 cartridge has a 0.308-inch-diameter bullet propelled by 30 grains of smokeless powder. As newer forms of gunpowder were developed, this powder charge was no longer used, but the terminology persists to this day. Additional misperceptions regarding caliber exists because the North Atlantic Treaty Organization (NATO) and US military projectiles are described using the metric system (7.62-mm or 9.00-mm rounds), whereas US civilian firearm munitions are generally referred to in measurements relating to inches (0.357 or 0.38). Unfortunately, no uniform mechanism exists for the description of firearm cartridges and manufacturers continue to inundate the market with further descriptions to add to the confusion, such as velocity, country of manufacture, number of grains of propellant, year of manufacture, and so forth (Fig. 3). As noted earlier, the question regarding caliber is commonly asked in the management of ballistic injury. In reality, caliber has minimal practical impact in the care of the patient, as the surgical management of a wound caused by a 0.357 projectile is no different from a wound caused by a 9-mm round, and should be directed to the specific anatomic anomaly created by the



Fig. 3 The tremendous variety of caliber, projectile composition or construction, and variable volumes of propellant and casings available for the modern firearm. (From Powers DB, Delo RI. Maxillofacial ballistic and missile injuries. In: Fonseca RJ, Walker RV, Betts NJ, et al, editors. Oral and maxillofacial trauma. 4th edition. St Louis (MO): Elsevier Saunders; 2012; with permission.)

projectile, not the weapon used. Experienced surgical providers cannot accurately determine the caliber of a weapon by visual examination of the wound alone, and would never alter the required treatment based on the diameter of the projectile.

Handguns are handheld firearms, with a barrel length generally 10.5 inches or smaller, which usually fire projectiles of a lower velocity and caliber. Handgun injuries generally have a tendency to "push-away," or stretch soft tissues, including vessels or nerves, as opposed to avulsive loss. The characteristic low-velocity wound has a small, rounded, or slightly ragged entrance wound, causing fragmentation of teeth and bony comminution, often exhibiting no exit wound (Fig. 4A–C). If an exit wound does occur, it is generally slit-shaped or stellate. Rifles are long guns with barrel lengths generally longer than 24 inches. At distance, rifle wounds create a low-energy transfer similar to those seen with handguns. At close range, the wounding characteristics are different because of the increased potential injury associated with velocity and high-energy transfer (see Fig. 4D–H). The presence of an exit wound is usually found, which may be stellate and larger than the entry wound. The existence of avulsive soft/hard tissue wounds and significant fragmentation of the bone can be characteristic findings of rifle wounds. A shotgun is a long gun that may fire a single pellet, or numerous pellets, at a relatively low velocity. The gauge of the shotgun is classified as the number of lead balls/pellets placed together, equaling the interior diameter of the barrel, which would weigh 1 pound. For contact with close-range injuries, the effect of the gas that is discharged under pressure into the wound also needs to be considered. This scenario is extremely important in shotgun and improvised explosive blast wounds because of the higher degree of contamination and presence of propelled gas and shock waves. Powder gases are expelled from the muzzle of the weapon after combustion of the gunpowder and follow the projectile out of the barrel. When the muzzle of the weapon is in contact with the target, this can be an additional source of tissue displacement, injury, and thermal burning.

Shotgun pellet injuries essentially depend completely on the distance the weapon is from the target at the time of discharge. Sherman and Parrish devised a classification system to describe

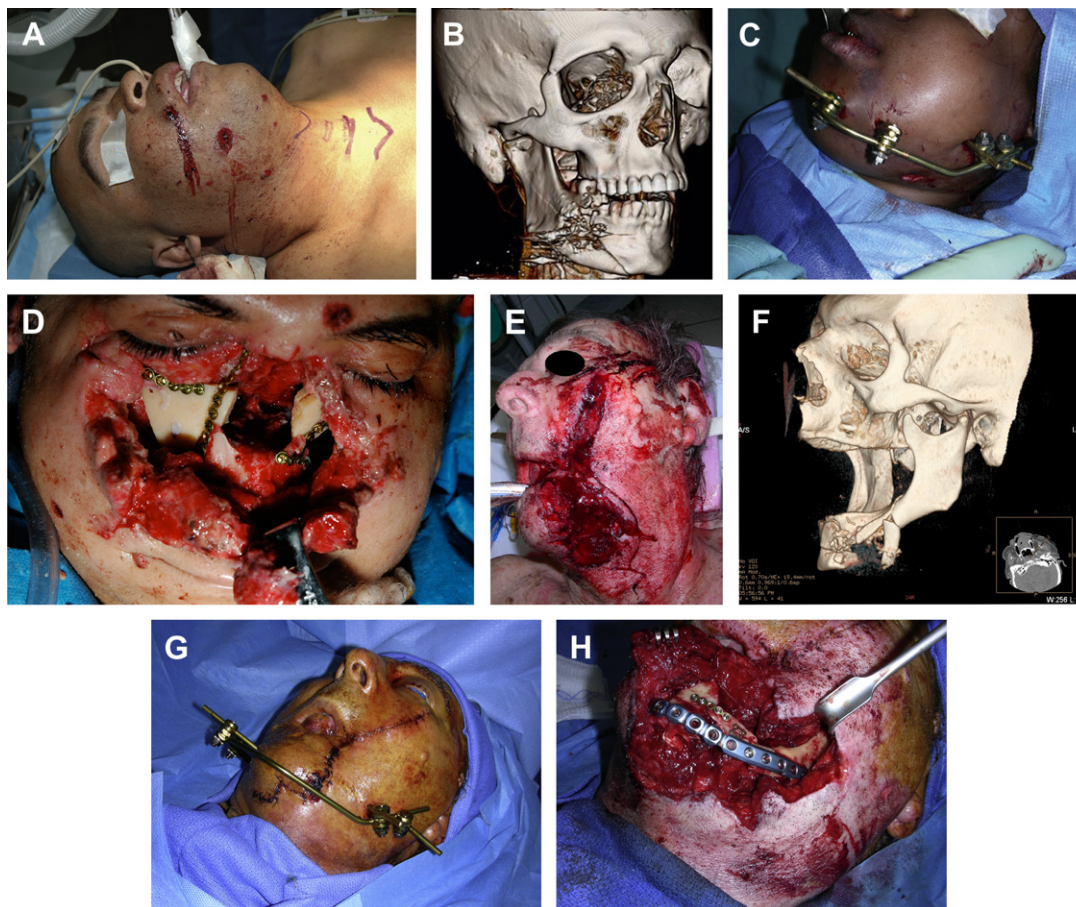


Fig. 4 (A) Characteristic clinical appearance of low-energy/low-velocity gunshot wound to the anterior mandible. No exit wound was detected. (B) Three-dimensional reconstruction of computed tomography scan indicating the degree of comminution associated with this gunshot wound. Three-dimensional reconstructions provide superior visualization, and localization, of anatomic variants in the management of ballistic injuries to the craniomaxillofacial unit. (C) Application of a modern external fixator for the management of a low-energy/low-velocity gunshot wound to the mandible. Note the conservative treatment of the gunshot wound, with minimal decontamination/debridement. (D) High-energy/high-velocity rifle wound to the anterior maxilla with complete avulsion of the nasal complex. Note the significant difference in the wounding characteristics of the high-energy weapon, as the patient was shot in the face at a distance by an assailant with a rifle. Reconstruction shows use of calvarial bone to reconstruct the vertical pillars of support for the maxilla. (E) High-energy gunshot wound to the anterior mandible. Note the presence of soft tissue disruption as the projectile exited the patient's mouth and then tracked along the soft tissues of his anterior maxilla. (F) Three-dimensional reconstruction of computed tomography scan indicating the degree of comminution and avulsive bone loss associated with this gunshot wound. (G) Initial stabilization of the patient was accomplished with an external fixator. (H) Definitive reconstruction with open reduction and internal fixation with a reconstruction plate. ([D] From Powers DB, Delo RI. Maxillofacial ballistic and missile injuries. In: Fonseca RJ, Walker RV, Betts NJ, et al, editors. Oral and maxillofacial trauma. 4th edition. St Louis (MO): Elsevier Saunders; 2012; with permission.)

shotgun wounds in relation to the distance from the target. Type I injury occurs from a distance longer than 7 yards; type II injury is sustained when the discharge is within 3 to 7 yards; type III injury is within 3 yards. Type III injuries usually sustain dramatic soft and hard tissue injuries and avulsion of tissue, whereas type I injuries may be minimal (Fig. 5). Because victims often have difficulty in determining how far away the shotgun was at the time of discharge, Glezer and colleagues revised this classification system and directed their attention to the size of the pellet scatter. Type I injuries occur when pellet scatter is within an area of 25 cm²; type II injuries are within 10 cm² to 25 cm²; type III injuries have pellet scatter less than 10 cm². Although the Glezer classification originally was developed for abdominal injuries, the information is transferable to other areas of the body, and determinations of tissue injury can be correlated directly to the size of the pellet scatter. Intuitively, the closer the shotgun is to the patient, the more dramatic is the hard and soft tissue

damage. For rifles and handguns, the practical clinical difference in whether the weapon was 10 feet, 100 feet, or 1000 feet away from the patient otherwise has no bearing on surgical and medical treatment.

Components of improvised explosive devices

The current conflicts in the Middle East have introduced a "new" mechanism for delivery of maxillofacial missile projectiles, resulting in gruesome and avulsive craniomaxillofacial injuries, the improvised explosive device (IED). Although not a new entity, as the concept of IEDs has been deployed by guerilla forces since World War II, the description and media interest in the IED warrants a brief discussion of its characteristic properties. Explosives are broadly classified as low-order explosives (LE, such as pipe bombs, gunpowder, or petroleum-based bombs) or

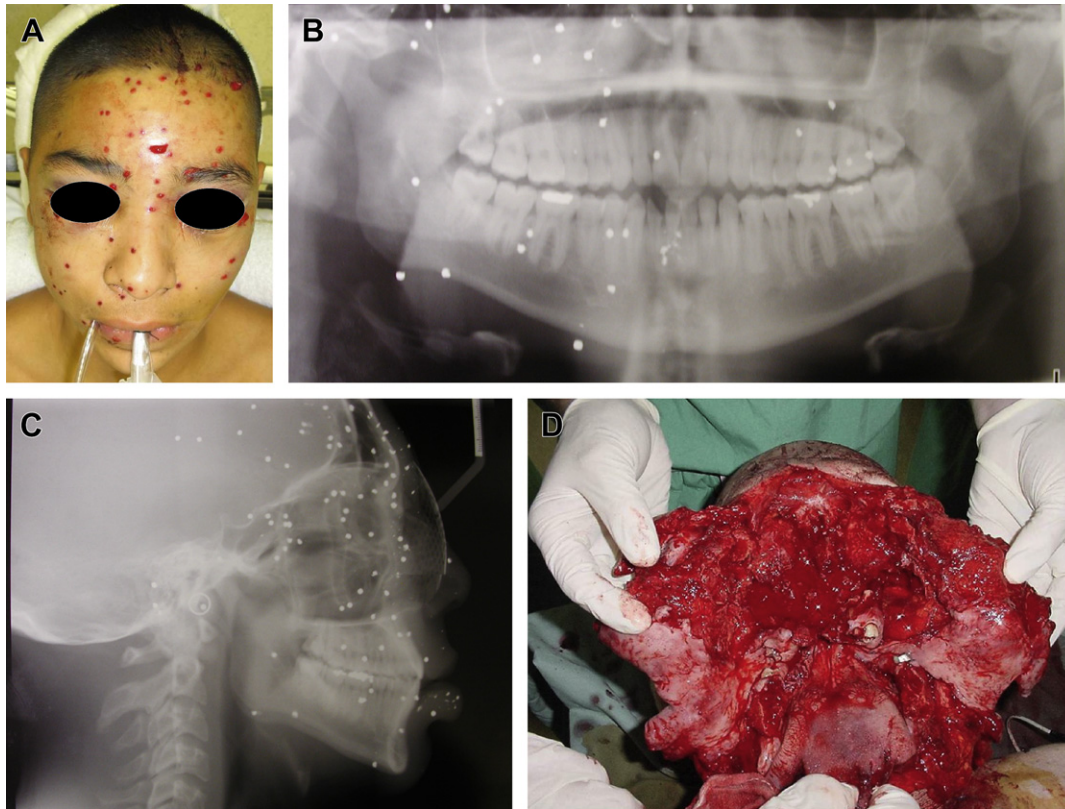


Fig. 5 (A) Characteristic facial appearance of a patient sustaining a shotgun wound from a distance (Sherman and Parrish – Class I or Glezer – Class I). Note the presence of multiple punctate entry wounds, but no significant disruption of the facial features. (B, C) Classic radiographic appearance of a patient sustaining a shotgun wound from a distance (Sherman and Parrish – Class I or Glezer – Class I). Note the presence of multiple shotgun pellets on the radiographs. (D) Self-inflicted shotgun wound in a suicide attempt. Note significant hard and soft tissue disruption and avulsion (Sherman and Parrish – Class III or Glezer – Class III). ([A, D] From Powers DB, Delo RI. Maxillofacial ballistic and missile injuries. In: Fonseca RJ, Walker RV, Betts NJ, et al, editors. Oral and maxillofacial trauma. 4th edition. St Louis (MO): Elsevier Saunders; 2012; with permission.)

high-ordered explosives (HE, such as TNT, C4, Semtex). Additionally, explosives are categorized as manufactured, which implies military-grade mass production and quality control, or improvised. An IED is a bomb fabricated in an “improvised” manner designed to destroy or incapacitate military personnel or civilians. The bomb itself may be a conventional military-grade weapon, or an assortment of explosive components, such as gasoline, or agricultural fertilizer, as seen in the Oklahoma City bombing of 1995. An IED has 5 components (Fig. 6):

- Switch (activator)
- Initiator (fuse)
- Container (body)
- Charge (explosive)
- Power source (battery)

Antipersonnel IEDs typically contain shrapnel, generating components such as nails, ball-bearings, metal fragments, wood, or glass. The victim may first sustain a burn injury from ignited explosives. Blunt and penetrating injury from contact by exploded fragments will further injure the patient. These fragments will be propelled at high or ultra-high velocity and therefore cause ultra-high kinetic energy injuries. Direct shrapnel injury is only a single element to be considered, as detonation of any powerful explosive generates a blast wave of high pressure that spreads out from the point of explosion and travels hundreds of yards in all directions. The relative

proximity of the victim to the site of the explosion, the greater the exposure to the shock wave energy. The initial shock wave of very high overpressurization, which is referred to as the primary, or “blast wave”, is unique to the HE and is followed closely by a “secondary wind,” a huge volume of displaced air flooding back into the area, again under pressure. It is these sudden and extreme differences in pressures, and associated

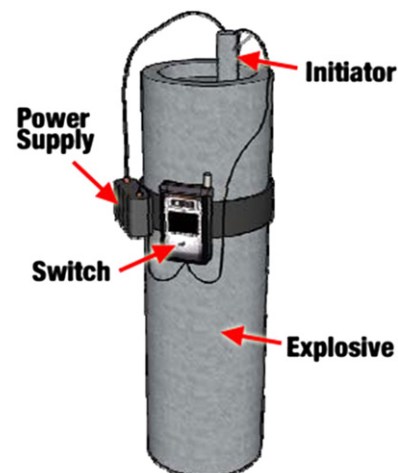


Fig. 6 Components of an IED.

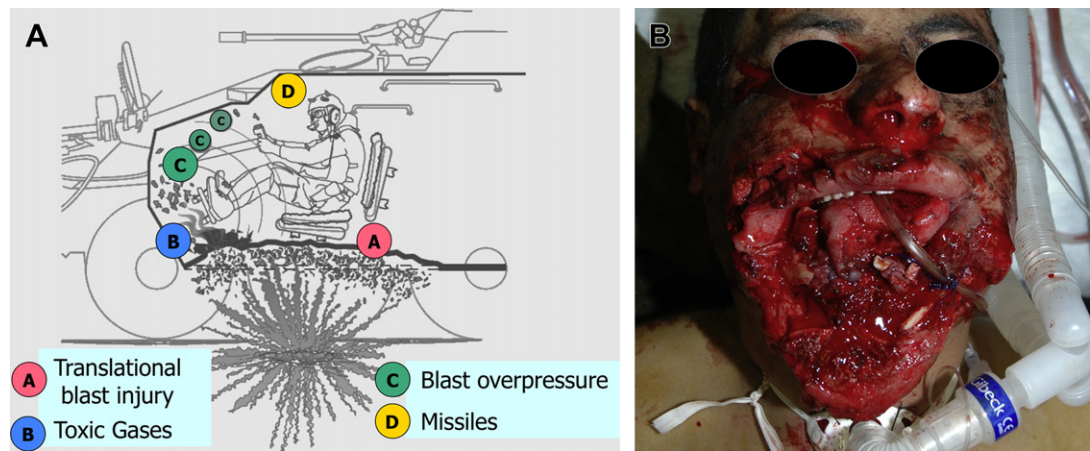


Fig. 7 (A) Wounding potential of an IED. (B) Characteristic facial injuries sustained by an improvised explosive device. ([A] From Emergency War Surgery Course. Washington, DC: US Government; 2009.)

dispersal of secondary projectiles, which can lead to significant neurologic, skeletal, or soft tissue injury (Fig. 7, Tables 2 and 3).

The principles of velocity

All else being equal, velocity has the largest impact on kinetic energy; however, velocity cannot be examined in a vacuum, as at suboptimal levels, expanding projectiles do not expand, and at excessive velocity, projectiles lose their stability in flight. The terms “high velocity” and “low velocity,” as they relate to projectiles, can also be somewhat misleading. Consensus between US and European research does not occur in the literature, with varying definitions correlating with where the study was performed (Tables 4–6). The US literature designates high velocity as being between 2000 and 3000 feet per second (610–914 m/s), whereas studies from the United Kingdom designate the line between low-velocity and high-velocity projectiles as being 1100 feet per second (335 m/s), which is the speed of sound in air. The earliest recognized entry of high-velocity projectiles having an association with increased

wounding potential occurred during the Vietnam War. In 1967, Rich reported in the *Journal of the American Medical Association* that bullets fired from the M16 rifle inflicted tremendous tissue destruction and injuries upon enemy combatants. The muzzle velocity of the projectile shot from the M16 was 3100 feet per second. When coupled with erroneous information published by Rybeck in 1974 and in the 1975 edition of the *Emergency War Surgery* manual regarding the size of the temporary cavity caused by the missile, this information led to the common misperception that high-velocity projectiles caused more significant injuries. Part of the confusion regarding the wounding potential of high-velocity projectiles is caused by misinterpretation of ballistic gelatin model studies. Ballistic gelatin is 10% to 20% gelatin refrigerated to 4° to 10°C and is used as the tissue model for ballistic studies. The wound-profile diagrams included in this article and others represent the findings of these studies. The validity of the ballistic gelatin model has been confirmed by comparison with human autopsies, although there is confusion in correlating these studies to living patients, because the human body is much more resistant to deformation than gelatin. The effects of skin resistance, clothing, and opposition to

Table 2 Mechanisms of blast injury			
Category	Characteristics	Body Part Affected	Types of Injuries
Primary	Unique to high-order explosives (HE), resulting from impact of the overpressurization wave with body surfaces	Gas-filled structures are most susceptible (lungs, middle ear, and gastrointestinal tract)	Pulmonary barotrauma (blast lung) Tympanic membrane rupture Abdominal hemorrhage and perforation Globe rupture Traumatic brain injury (TBI)
Secondary	Results from flying debris and bomb fragments	Any	Penetrating ballistic injury caused by shrapnel or fragmentation Blunt injuries Globe penetration (can be occult)
Tertiary	Results from individuals being thrown by the blast wind	Any	Fractures Traumatic amputation Open/closed TBI
Quaternary	All blast/explosion-related injuries/ illnesses/diseases not related to other mechanisms (includes exacerbations or complications associated with existing diseases)	Any	Burns Crush injuries Open/closed TBI Asthma/chronic obstructive pulmonary disease/Angina/Hypertension/Hyperglycemia

Table 3 Overview of improvised explosive device/blast-related injuries to the craniomaxillofacial region

System	Injury or Condition
Auditory	Tympanic membrane rupture
	Ossicular disruption
	Cochlear damage
Eye/Orbit/Face	Foreign body entrapment
	Perforated/Penetrated globe
	Foreign body entrapment
	Air embolism
	Fractures
	Thermal injury
Respiratory	Soft tissue disruption
	Blast Lung: direct consequence of the high-order explosive overpressurization wave.
	The most common fatal primary blast injury among initial survivors
	Hemothorax
	Aspiration pneumonitis
	Airway thermal injury
CNS Injury	Pulmonary contusion/hemorrhage
	Open/closed traumatic brain injury
	Cerebrovascular accident
	Spinal cord injury
	Air embolism—induced injuries

separation of the fascial planes cannot be replicated in gelatin. Harvey evaluated the 2 types of pressure waves produced by penetrating objects in 1947: the sonic pressure wave and the temporary cavity. The first wave is the sonic pressure wave, sometimes referred to as the "shock wave," and it relates the sound of the projectile striking the target. This wave transmits at the speed of sound (ie, approximately 4750 feet per second [1450 m/s]) and is traveling considerably faster than the projectile entering the target. No temporary cavity is formed with the sonic pressure wave, and in that regard it is analogous to the lithotripsy devices used for renal calculi destruction, with corresponding minimal risks for tissue injury. Although American and Swedish researchers have tried to disprove Harvey's conclusions, no definitive evidence suggests that his findings are in error, and additional studies by French and American researchers support the original findings of 1947.

The secondary pressure wave, referred to as the temporary cavity, is formed when the penetrating projectile strikes tissue and the wave radiates away laterally away from the permanent cavity of the projectile path. After being struck by the projectile, the ballistic gelatin/tissue displays an obvious temporary cavity, which potentially injures tissues, such as muscle, vessels, and organs. The clinical significance of this cavity is variable, with no real consensus in the literature, and the temporary cavity caused by the M16 in animal laboratory models is much smaller than the approximate 18-cm temporary cavity seen in ballistic gelatin. Dog models indicated that acute tissue

Table 4 Ballistic table for common handgun cartridges

Cartridge	Velocity (fps)—muzzle	Velocity (fps)—100 yd	Energy (fpe)—muzzle	Energy (fpe)—100 yd
0.25	900	742	63	43
0.32	1000	834	133	96
0.38	800	735	199	168
9 mm	975	899	310	264
0.357 Magnum	1500	1153	624	298
0.44 Magnum	1500	1196	999	635
0.45	970	860	386	304
0.50 Magnum	1700	1289	2246	1291

Table 5 Ballistic table for common rifle/machine gun cartridges

Cartridge	Velocity (fps)—muzzle	Velocity (fps)—100 yd	Energy (fpe)—muzzle	Energy (fpe)—100 yd
0.22 Hornet	3070	2246	732	392
0.243	3010	2744	1911	1588
0.270	3060	2851	2702	2345
0.30–30	2390	1959	1902	1278
0.30–06	2960	2750	3209	2769
5.56 mm (NATO)	2910	2675	1410	1192
7.62 mm (AK 47)	2360	2060	1521	1159
9-mm Parabellum (Uzi)	1060	946	338	268
0.50 BMG Sniper	2820	2732	13421	12428

Abbreviations: fpe, foot-pounds of energy; fps, feet per second.

Table 6 Ballistic table for common shotgun slugs

Cartridge (2.75-in. shell)	Velocity (fps)—muzzle	Velocity (fps)—100 yd	Energy (fpe)—muzzle	Energy (fpe)—100 yd
12-gauge (1-oz slug)	1560	977	2364	927
16-gauge (0.9-oz slug)	1590	975	2320	875
20-gauge (0.87-oz slug)	1590	975	2080	780

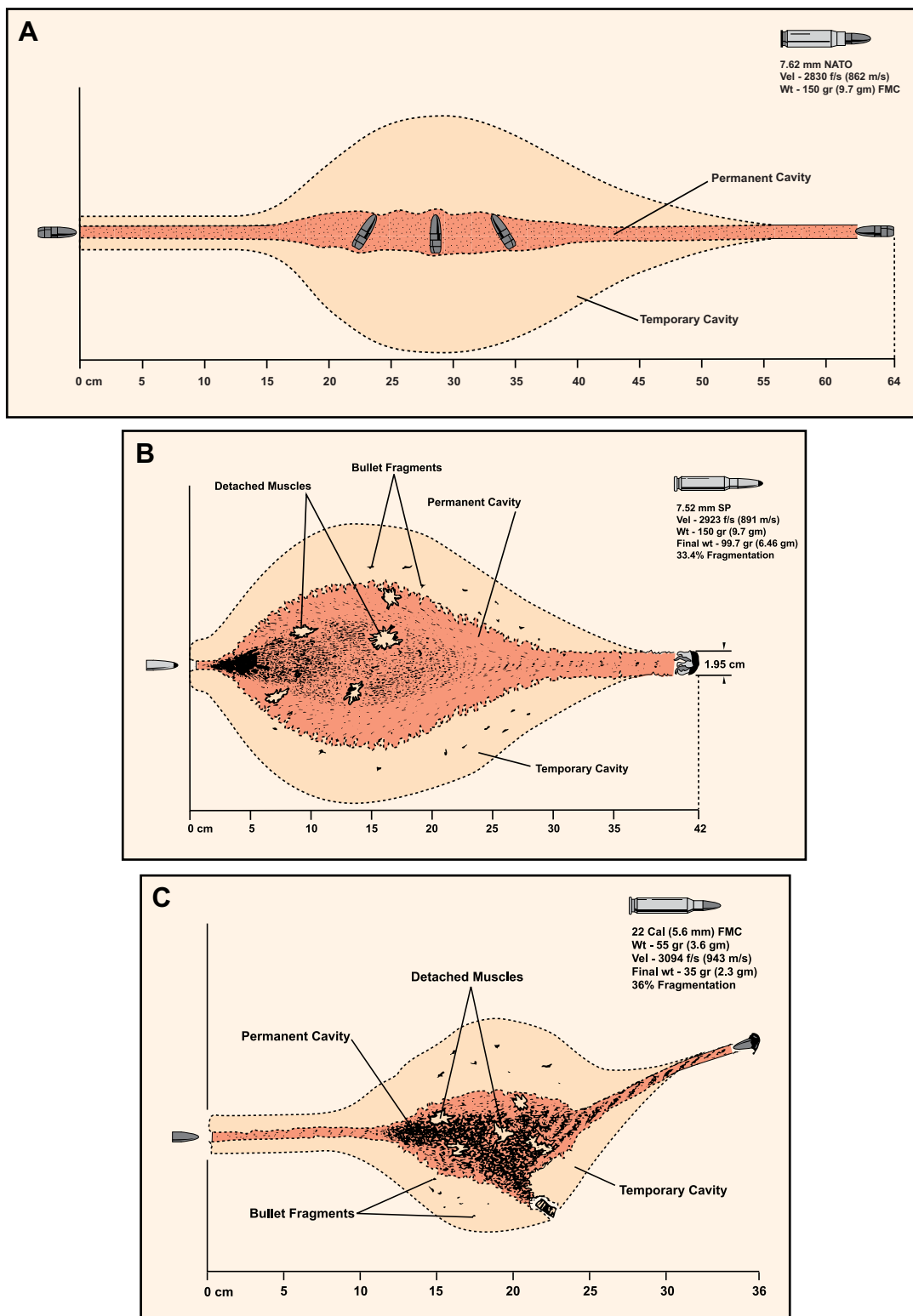


Fig. 8 (A) Ballistic representation of NATO 7.62-mm round fired from M16 rifle. Observe the relatively consistent permanent cavity and laterally radiating temporary cavity, which begins to develop at approximately 20 cm into the tissue as the projectile begins to tumble. This chart represents the projectile not striking any hard structures causing deformation or alteration in trajectory. The anatomic characteristics of the head and neck do not have more than 20 cm of soft tissue present before encountering the bony skeleton, which would have a clinical significance with regard to the temporary cavity should the projectile be of a trajectory to encounter only soft tissue and miss the underlying facial bones. (B) Ballistic representation of a 7.52-mm soft point (SP) round striking muscle and bone. Note as the projectile strikes the underlying structures, there is a tremendous increase in the permanent cavity, as well as the temporary cavity, as the projectile deforms and fragments because of the soft tip construction. This deformation in the structural characteristic of the projectile, and associated increase in the permanent and temporary cavities, greatly enhances the wounding potential of this round. (C) Ballistic representation of a 22-caliber (5.6-mm) full-metal case (FMC) round striking bone and muscle. Note as the relatively small caliber projectile strikes the underlying structures, there is a tremendous increase in the permanent cavity and associated temporary cavity as the projectile deforms and continues on a new trajectory. This representation illustrates the wounding potential of a smaller caliber weapon should the projectile actually strike the target and engage in energy transfer to the tissues. (From *Emergency War Surgery*. 3rd US Revision. Washington, DC: US Government Printing Office; 2004.)

injury secondary to temporary cavity formation sustained with high-velocity projectile strikes were no more than 5 cm and were able to resolve within 72 hours. The US military conducted extensive research into the wounding patterns of projectiles, and the results are summarized in Fig. 8. The unique anatomic differences of the craniomaxillofacial skeleton, a relatively thin soft tissue layer overlying a dense foundation of bone, mitigate some of the expected responses of the temporary tissue stretch, as the overall thickness of the soft tissue envelope is generally less than the required total distance needing to be traveled before exhibiting secondary cavitation. Although sequential soft tissue necrosis and small-vessel damage can occur, it is much more likely to be in response to the exaggerated permanent cavity of the projectile, which is greatly enhanced after striking the underlying facial skeleton. The key point of understanding in the management of ballistic injuries is the permanent cavity, which involves all of the tissues that are pushed aside or destroyed during the flight of the projectile, and is the location of the extent of the initial, or immediate, damage. A projectile striking bone may cause fragmentation of the bullet and/or native bone, forming numerous secondary missiles, each capable of producing additional wounds, dramatically increasing the size of the permanent cavity (Fig. 9). The size and shape of the permanent cavity are determined by the density and anatomic characteristics of the tissue lying in the projectile's path, the velocity of the projectile, the shape/characteristics of the projectile, and likely most importantly the degree of deformation of the missile as it travels through the tissues.

Characteristics of ballistic injuries

Gunshot injuries have been categorized in the literature as penetrating, perforating, or avulsive. Penetrating wounds are caused by the projectile striking the victim but not exiting the body. The perforating injuries have entrance and exit wounds, classically described as being without appreciable tissue loss. Avulsive injuries have entrance and exit wounds, generally presenting with an acute loss of tissue associated with the passage of the projectile out of the victim. The type of firearm used has implications in the wounding potential of the projectile. As referenced earlier in this article, traditional concepts of ballistics teach that impact kinetic energy is equal to one-half the mass of the projectile times velocity squared ($KE = \frac{1}{2} MV^2$), the increased energy transmitted from a high-velocity projectile does not necessarily translate to increased wounding capacity.



Fig. 9 Example of a projectile striking the mandible, causing fragmentation of the bone with the formation of numerous secondary projectiles, which enlarged the size of the permanent cavity.

Cunningham and others suggest modifications need be used to correct the kinetic energy estimate of wounding potential for the type of tissue being struck by the projectile. Cunningham's belief was that softer tissues, such as brain and muscle, should be associated with a lower exponent of injury (0.5) than harder tissues, such as bone, which would have a higher exponent (2.5) and therefore higher likelihood of permanent injury. The corrected formula for estimating wounding capacity by kinetic energy should be $KE = \frac{1}{2} MV^{0.5}$ to $KE = \frac{1}{2} MV^{2.5}$.

The soft tissue injuries inherent in ballistic trauma may exhibit avulsive loss, sequential necrosis over days to weeks, and compromised vascularity negating, or delaying, potential microvascular or pedicled soft tissue reconstruction. Because of the frequent occurrence of comminuted bony fractures, the necessity for open reduction of the hard tissue injuries further complicates the soft tissue healing response. A compromised soft tissue bed can lead to necrosis of free-floating bone fragments, avascular necrosis of the underlying facial skeleton, devitalization of stabilized fracture segments, and development of soft tissue infection or osteomyelitis, resulting in increased tissue loss and scarring of the facial composite. Hard tissue loss, including both bone and teeth, present the unique challenges of reconstruction, including reconstitution of the masticatory complex to support the oral intake of nutrition, reestablishment of the normal anterior-posterior projection and angular shape of the facial skeleton, maintenance of lip competence, and control of salivation. Beyond the anatomic concerns of reconstruction, the presence of specialized vascular and neurosensory components in the maxillofacial region, including the great vessels of the neck, the various branches of the cranial nerves compromising both motor and sensory functions, such as sight, smell, hearing, and taste, only serve to further complicate the potential for catastrophic injury, and lifelong deformity, that ballistic injuries cause to the craniomaxillofacial region.

Summary

Ballistic injury wounds are formed by variable interrelated factors, such as the nature of the tissue, the compositional makeup of the bullet, distance to the target, and the velocity, shape, and mass of the of the projectile. This complex arrangement, with the ultimate outcome dependent on each other, makes the prediction of wounding potential difficult to assess. As the facial features are the component of the body most involved in a patient's personality and interaction with society, preservation of form, cosmesis, and functional outcome should remain the primary goals in the management of ballistic injury. A logical, sequential analysis of the injury patterns to the facial complex is an absolutely necessary component for the treatment of craniomaxillofacial ballistic injuries. Fortunately, these skill sets should be well honed in all craniomaxillofacial surgeons through their exposure to generalized trauma, orthognathic, oncologic, and cosmetic surgery patients. Identification of injured tissues, understanding the functional limitations of these injuries, and preservation of both hard and soft tissues minimizing the need for tissue replacement are paramount.

Further readings

Barach E, Tomlanovich M, Nowak R. Ballistics: a pathophysiologic examination of the wounding mechanisms of firearms. Part I. *J Trauma* 1986;26:225.

- Barnes FC. Cartridges of the world: a complete and illustrated reference for over 1500 cartridges. 12th edition. Iowa (WI): F+W Media Inc; 2009.
- Clark N, Birely B, Manson PN, et al. High-energy ballistic and avulsive facial injuries: classification, patterns, and an algorithm for primary reconstruction. *Plast Reconstr Surg* 1996;98(4):583–601.
- Cunningham LL, Haug RH, Ford J. Firearm injuries to the maxillofacial region: an overview of current thoughts regarding demographics, pathophysiology and management. *J Oral Maxillofac Surg* 2003;61:932–42.
- Di Maio VJ. Gunshot wounds: practical aspects of firearms, ballistics, and forensic techniques. 2nd edition. Washington, DC: CRC Press; 1999. p. 16–27.
- Explosions and blast injuries: a primer for clinicians. National Center for Injury Prevention and Control, Centers for Disease Control and Prevention. Available at: <http://www.bt.cdc.gov/masscasualties/explosions.asp>. Accessed September 1, 2012.
- Fackler ML, Bellamy RF, Malinowski JA. The wound profile: illustration of the missile-tissue interaction. *J Trauma* 1988;28:S21.
- Fackler ML. Civilian gunshot wounds and ballistics: dispelling the myths. *Emerg Med Clin North Am* 1998;16:17–28.
- Fackler ML. Gunshot wound review. *Ann Emerg Med* 1996;28:194–203.
- Fackler ML. The wound and the human body: damage pattern correlation. *Wound Ballistics Review* 1994;1:12–9.
- Glezer JA, Minard G, Croce MA, et al. Shotgun wounds to the abdomen. *Am Surg* 1993;59:129.
- Harvey EN, Korr IM, Oster G, et al. Secondary damage in wounding due to pressure changes accompanying the passage of high velocity missiles. *Surgery* 1947;21:218–39.
- Ordog GJ, Balasubramanian S, Wasserberger J, et al. Extremity gunshot wounds. I. Identification and treatment of patients at high risk of vascular injury. *J Trauma* 1994;36:358–68.
- Ordog GJ, Wasserberger J, Balasubramanian S. Wound ballistics: theory and practice. *Ann Emerg Med* 1985;13:1113.
- Powers DB, Will MJ, Bourgeois SL, et al. Maxillofacial trauma treatment protocol. *Oral Maxillofac Surg Clin North Am* 2005;17:341–55.
- Rich NM, Johnson EV, Dimond Jr FC. Wounding power of missiles used in the Republic of Vietnam. *JAMA* 1967;199:157–61.
- Robertson BC, Manson PN. High-energy ballistic and avulsive injuries: a management protocol for the next millennium. *Surg Clin North Am* 1999;79(6):1489–502.
- Rybeck B. Missile wounding and hemodynamic effects of energy absorption. *Acta Chir Scand* 1974;450(Suppl):5–32.
- Sherman RT, Parrish RA. Management of shotgun injuries: a review of 152 cases. *J Trauma* 1963;3:76.
- Suneson A, Hansson HA, Lycke E, et al. Pressure wave injuries to rat dorsal root ganglion cells in culture caused by high-energy projectiles. *J Trauma* 1989;29:10–8.
- Suneson A, Hansson HA, Seeman T. Central and peripheral nervous system damage following high-energy missile wounds in the thigh. *J Trauma* 1988;28(Suppl 1):S197–203.
- Suneson A, Hansson HA, Seeman T. Pressure wave injuries to the nervous system caused by high-energy missile extremity impact. I. Local and distant effects on the peripheral nervous system: a light and electron microscopic study on pigs. *J Trauma* 1990;30:281–94.
- Tan YH, Zhou SX, Liu YQ, et al. Small-vessel pathology and anastomosis following maxillofacial firearm wounds: an experimental study. *J Oral Maxillofac Surg* 1991;49(4):348–52.
- United States Government Printing Office. Emergency war surgery. Third United States Revision. Washington, DC: United States Government Printing Office; 2004.
- Ziervogel JF. A study of the muscle damage caused by the 7.62 NATO rifle. *Acta Chir Scand Suppl* 1979;489:131.

Maxillofacial Imaging in the Trauma Patient

Gerald T. Grant, DMD, MS ^{a,b,*}, Peter Liacouras, PhD ^a, Shayne Kondor, BAE, MAE ^b

KEYWORDS

• Craniofacial imaging • Virtual surgery • Medical models • DICOM

KEY POINTS

- Recent advances in imaging systems, software, and the use of additive manufacturing techniques have provided unprecedented opportunities for evaluation of both hard and soft tissues for presurgical planning, custom implant fabrications, and surgical guides.
- Standardization of the medical image files as digital imaging and communications in medicine (DICOM) has made possible the development of software that can evaluate, manipulate, and reformat medical images that make them easily converted to file formats to fabricate medical models and custom devices from industrial manufacturing processes.
- These standards are applied to all medical capture devices to include magnetic resonance, ultrasound, conventional computed tomography (CT) scans, and dental cone beam CT (CBCT) ensuring that all of these files are compatible with picture archiving and communication systems, which store and provide access from multiple modalities.

Cephalometric radiographs, pantographic radiographs, and computed tomography (CT) have historically been used for imaging for reconstruction of the craniofacial structures. The development of 3-dimensional (3D) rendering algorithms of the viewing software for medical images provided the information to allow surgeons the ability to better visualize boney and soft tissue defects. In addition, standardization of the medical image files as digital imaging and communications in medicine (DICOM) has made possible the development of software that can evaluate, manipulate, and reformat medical images that make them easily converted to file formats to fabricate medical models and custom devices from industrial manufacturing processes. These standards are applied to all medical capture devices to include magnetic resonance, ultrasound, conventional computed tomography (CT) scans, and dental cone beam CT (CBCT) ensuring all of these files are compatible with picture archiving and communication systems (PACS), which store and provide access from multiple modalities. Recent advances in imaging systems, software, and the use of additive manufacturing techniques have provided unprecedented opportunities for evaluation of both hard and soft tissues for presurgical planning, custom implant fabrications, and surgical guides.

The goal of this article is to introduce the following concepts and how they provide craniofacial reconstruction:

- Radiographic imaging systems used in head and neck reconstruction
- 3D surface imaging (stereophotogrammetry)
- 3D software reconstructions and virtual surgical techniques
- Custom reconstruction techniques

Radiographic images

CT

CT generates internal and external anatomic views by a radiographic approach (Fig. 1). A 1-dimensional (slice or fan-shaped) beam of x-ray radiation is directed through the subject. Radiodense tissues block some of the x-ray photons, and the transmitted photons are detected on the opposite side of the

subject. The x-ray emitter and x-ray detector oppose each other on a rotating ring. As the ring rotates around the subject, a 1-dimensional (1D) radiographic dataset is obtained. The 1D dataset is mathematically transformed into a 2-dimensional (2D) radiographic view in the plane of the ring. This 2D view is a single slice of finite thickness. The ring is axially traversed to a new location along the long axis of the subject and the process is repeated to obtain additional 2D slice images. A set of 2D sliced images normal to a traversing axis, taken at known locations, forms a tomographic dataset. The tomographic dataset can be visualized in 3 dimensions, showing a view of the internal anatomy in reference to the external anatomy, as shown in the accompanying image (Fig. 1). In a typical CT slice image of the head, bone is easily discerned from soft tissue, where high radiodensity tissues display brighter than lower radiodensity tissues, and air displays black (Fig. 2).

Tomographic slices are stored as digital grayscale images. Each pixel has a calibrated, known size; the matrix of pixels defines the area covered by the slice. The grayscale value of each pixel is encoded to the x-ray radiodensity of the tissue at

^a Department of Radiology, 3D Medical Applications Center, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, MD 20889, USA

^b Craniofacial Imaging Research, Navy Medical Personnel Training Center, Naval Postgraduate Dental School NMPTC, 8901 Wisconsin Avenue, Bethesda, MD 20889, USA

* Corresponding author. 3D Medical Applications Center, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, MD 20889.

E-mail address: Gerald.t.grant.mil@health.mil



Fig. 1 3D Reconstruction of CT scan data.

that spatial location. Grayscale values in medical CT images are calibrated to the Hounsfield Unit scale (HU), where distinct tissue types have definite HU value ranges.¹ A tomographic slice represents a finite thickness slice through the anatomy, and the spacing between each image slice is known; thus, each pixel can be assigned a thickness to become a 3D element, or a voxel. Medical CT voxels are generally orthotropic, being taller than the width or depth.

Axial slice sets can be reformatted and displayed as coronal, sagittal, or oblique slice views by displaying the projection of voxels along any plane of interest. Fig. 3 shows a combination of orthogonal views generated from an axial tomographic dataset. In cases of large slice distances, orthotropic voxels may cause the image to appear distorted or saw-toothed in transverse projections.²

Three-dimensional views of the anatomy are generated assigning color and opacity values to the voxels and displaying

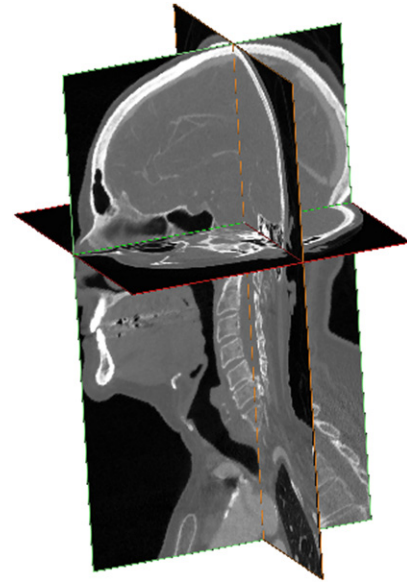


Fig. 3 Combination of views from an axial tomographic dataset.

the projection along a defined viewing direction. In the volumetric rendering, tissues are visually contrasted by unique color and opacity values assigned based on the HU value of the voxel (Fig. 4). Individual anatomic structures can be segmented from the dataset by grouping and displaying only voxels in a desired HU values range. Typically, hard tissue structures are readily differentiated from soft tissue, but soft tissue types are not as readily differentiated.²

Radiopaque objects in the field of the CT slice will cause streak artifacts in the image (Fig. 5). Dental restorations, dental implants, bone-mending plates, screws, vascular clips, and shrapnel can cause streaks in the image, which occlude the view of the anatomy. However, these artifacts are limited to slice plane(s) containing the radiopaque objects. Metal artifact reduction algorithms are often applied during the construction of the slice image to reduce distortion. Careful segmentation of a CT scan of a cadaver skull with metal spheres demonstrates



Fig. 2 Typical slice from a CT scan.

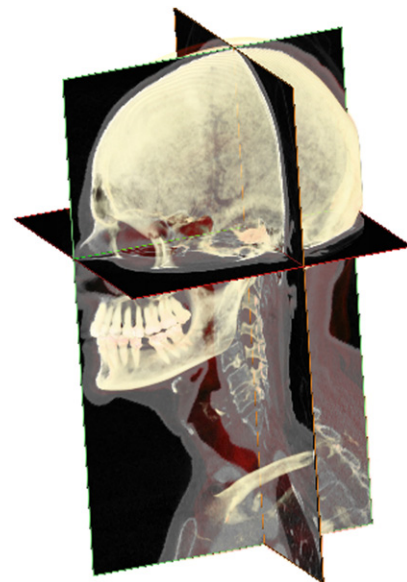


Fig. 4 Volumetric views contrasted in color.

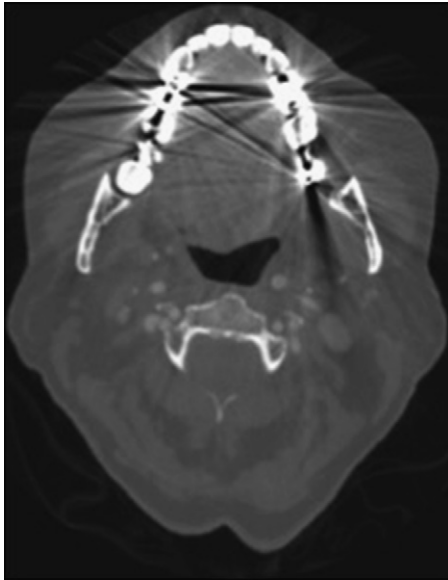


Fig. 5 Streak artifact in CT axial slice image.

that the metal artifacts can be minimized in the 3D model construction. Small streak artifacts can still be noted on the sphere near the mastoid process and on the posterior of the skull (Fig. 6).

Medical CT scan radiation exposure to tissues of the head can be significant. Radiation dose for a maxillofacial scan has been reported in the range of 534 to 860 μSv effective dose.³

CBCT

CBCT is an alternate radiographic imaging approach to obtain a tomographic dataset of the head. This modality is typically used to support dental treatment, especially in planning dental implant placement.

CBCT differs from CT in that the entire volume of the field of view is captured in a single rotation of the x-ray emitter and detector around the subject. In this case, multiple 2D x-ray image projections are collected, at radial intervals around the head. The set of 2D images are mathematically transformed to

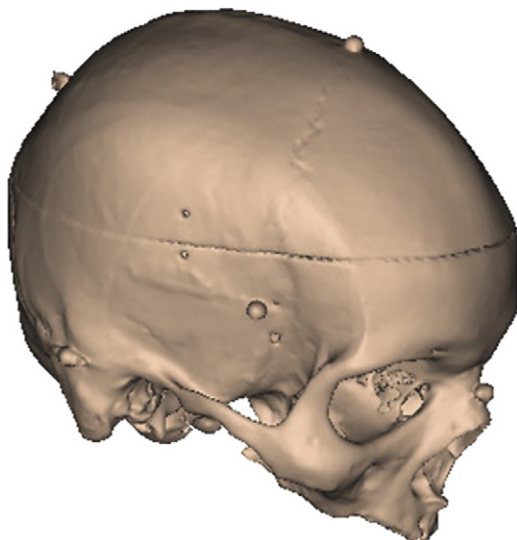


Fig. 6 Reconstructed 3D model from CT scan.

a 3D voxel data set. The volume data are then stored as a set of 2D slices images at regular intervals.

A direct comparison of CBCT to multidetector CT (MDCT) image quality can be seen in Figs. 7 and 8, each from approximately the same axial location on the same subject; Fig. 7 is from a large-field CBCT scan, whereas Fig. 8 is from an MDCT scan. Contrast between tissue structures is generally lower in the CBCT scan. Furthermore, it can be noted that the gray value of bone is inconsistent, varying with location in the CBCT slice. The latter effect is caused by beam-hardening artifact.^{2,4} Because of the severity of the beam-hardening effect, the CBCT cannot be calibrated to the Hounsfield Unit Scale.

Disadvantages of CBCT:

- Metal streak artifacts and image noise are generally more severe in CBCT scans. Fig. 9 shows the effect of multiple dental restorations in a CBCT scan slice. The radiating light and dark streaks are similar to the medical CT image, except the starburst effect is present throughout the image. Fig. 10 shows similar effects from a bone-anchored hearing aid (in the lower left corner of the slice).
- Truncation artifacts, or streaks radiating from the edges of features with differing radiodensity, are very prevalent in CBCT images.
- A gray value noise similar to analog television static is also prevalent in the images.

Advantages of CBCT:

- CBCT images have an advantage of high resolution, generally higher pixel resolution than most medical CT devices.
- CBCT slice thickness is generally identical to the pixel dimensions resulting in cubic (or isotropic) voxel. Isotropic data can be reformatted along any plane with minimal distortion in the image.
- CBCT scans are inexpensive and take only about 30 to 60 seconds to complete.
- The CBCT generally has the advantage of a significantly lower radiation dose for a maxillofacial scan, in the range of 70 to 98 μSv effective dose³ depending on the device; however, a few outlier devices can rival the radiation dose reported for an MDCT scan.



Fig. 7 Large-field CBCT scan.



Fig. 8 Slice of CT scan.

Magnetic resonance imagery

Magnetic resonance images (MRI) are an imaging modality that provides an internal view of the anatomy without exposing the subject to x-ray radiation. Images are generated from the reaction of hydrogen atoms in tissues interacting with a strong magnetic field. The greater the number of hydrogen atoms present, the stronger the induced signal from that tissue.

Magnetic field changes caused by the moving hydrogen atoms induce currents in a receiver coil. Similar to the CT scan principle, 1D data from the coil is transformed into a 2D slice, and encoded as gray value pixels in a digital image matrix (Figs. 11 and 12).

Disadvantages:

- Bone does not provide a return signal.
- High-resolution scans take a long time and are usually degraded by subject movement.
- Slice thickness and spacing are generally much larger than CT or CBCT scans.
- Coil sensitivity is not uniform; grayscale values can shift from position to position in the image.

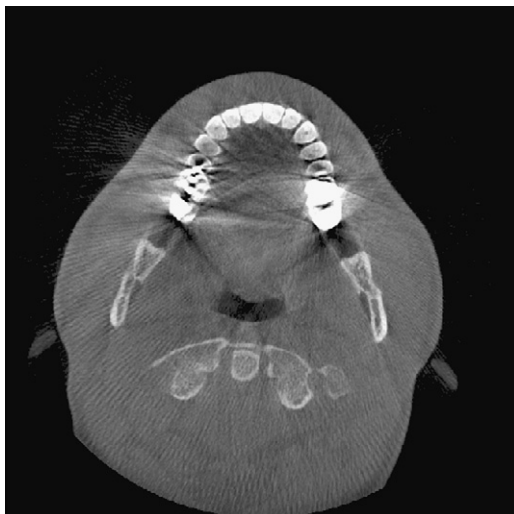


Fig. 9 Dental restoration artifact in CBCT.

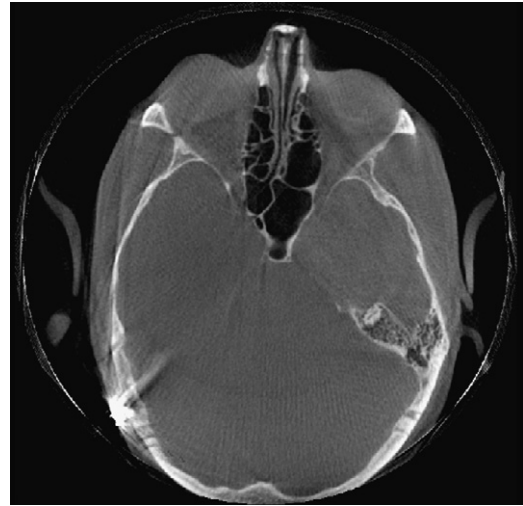


Fig. 10 Streak artifact from bone-anchored device in CBCT.

- Images can be distorted by a number of artifacts resulting from radiofrequency radiation patterns.
- Cannot scan patients with ferrous metal implants.

Advantages:

- No radiation exposure to the patient
- High slice plane resolution
- Excellent soft tissue differentiation

Surface images

Optical scanning (3D Photography)

The use of multiple cameras to capture 3D information has become more available through many different commercial systems. Optical scanning can be 2 or more cameras that capture data at different angles that are interpreted by software to provide a 3D image. Many of the file forms are in the

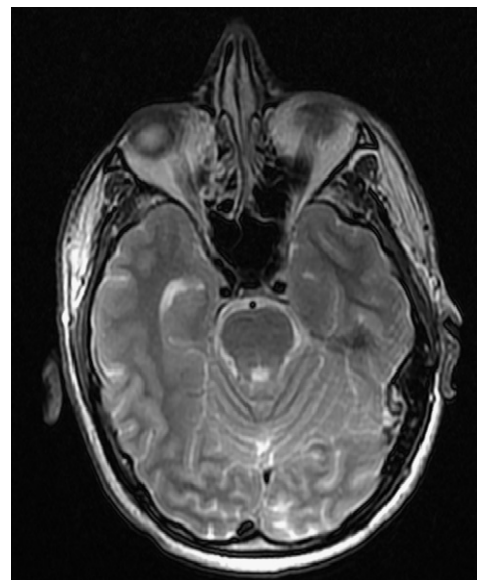


Fig. 11 MRI scan at slice of cranial vault.

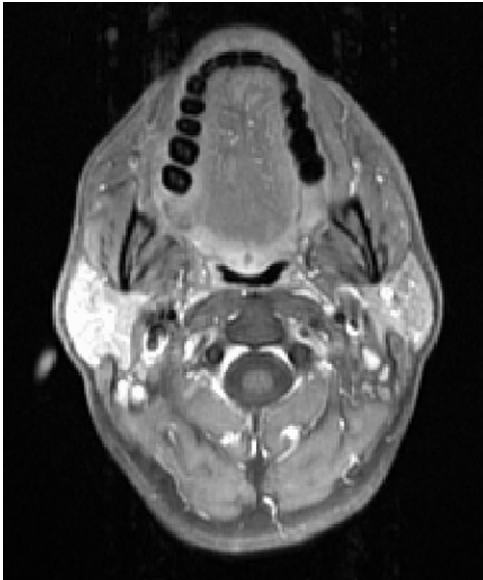


Fig. 12 MRI scan at the slice to include the cranial base.

format of an .stl (standard tessellation language).⁵ Fig. 13 shows a 5-camera system that can capture the entire head. Fig. 14 is an example of an image that is captured. These images can provide information as to the surface of the soft tissue, and can be registered to both CBCT and CT scans (Fig. 15). This type of information is helpful for virtual surgical manipulation in that it can better provide visualization as to the soft tissue reactions to bony movements within the craniofacial treatment plan.

Laser scanning

Lasers are also used to capture the surface of the soft tissue, and are often used in the scanning of residual limbs for prosthetic devices. Generally because of the need to place registration markers, interference with magnetic third point of references, or the time that it takes to make the scan which can introduce movement, it is not as popular of a technique for surface capture as the photoscanners.



Fig. 13 Five-camera photo camera system to capture the entire head as a digital image.



Fig. 14 Image from photo-capture with the color mapping in place.

Reconstruction of digital images

Digital software can import standard DICOM images and allows for tissue segmentation based on contrast or HUs. The result is a highly accurate patient-specific digital reconstruction of the bony or soft tissue geometry (Fig. 16). A multitude of digital operations can be performed using these programs to obtain the exact anatomic structures needed. Software can then be used to develop a 3D computer model providing the reconstruction team the ability to visualize the defects that could not be generated from standard radiographic techniques. These images can be then modified to develop reconstruction plans; surgical guides and custom fixation devices can be designed to enable the plan. The digital files of the computer model, surgical guides, and custom devices are then exported as an .stl file, a triangular mesh-type structure, which can then be manufactured on a multitude of additive manufacturing machines.

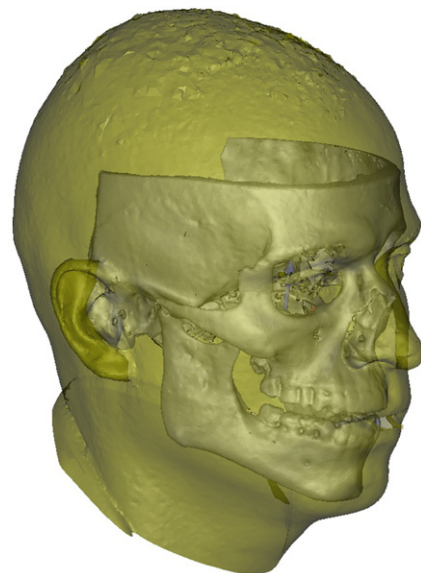


Fig. 15 Soft tissue image from a photo-captured image registered to a CBCT of the patient.

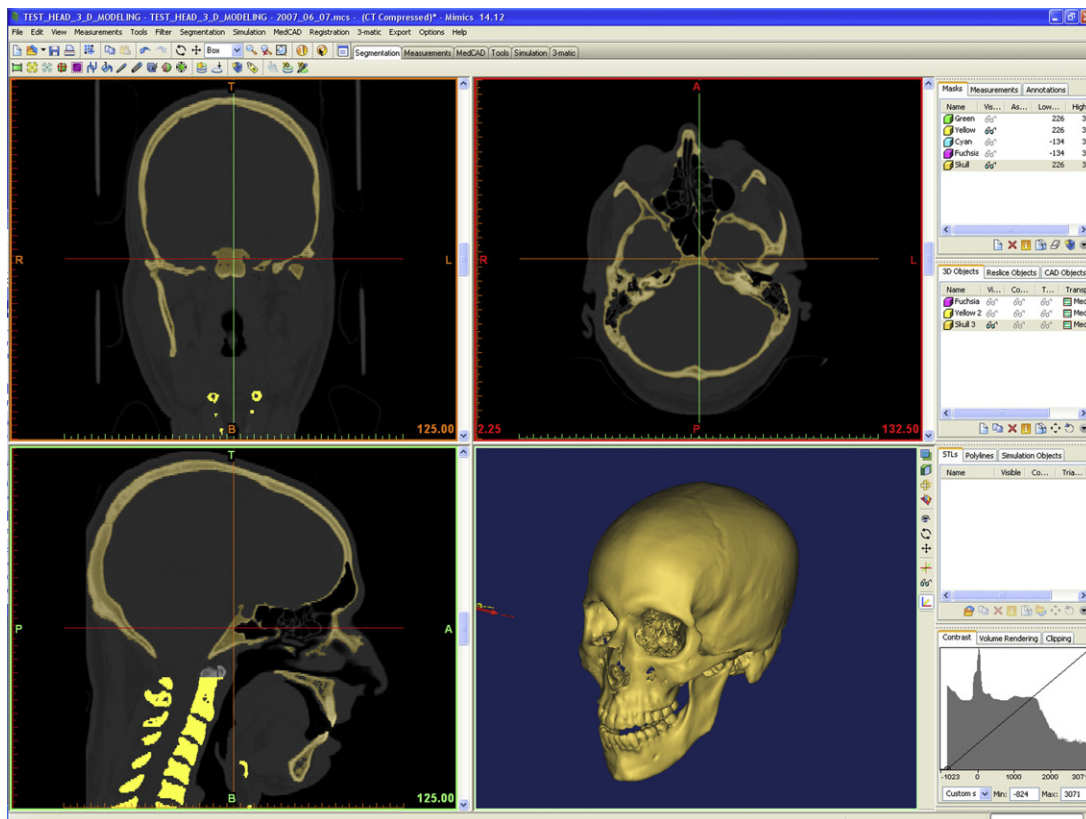


Fig. 16 Software 3D reconstruction from CT scan.

Normal CT images versus anatomic model

The CT of a midface injury (Fig. 17) contains information that includes the radiologist's notes that describe this patient as

having multiple comminuted fractures through the maxilla and across many sections of the orbital and nasal regions. The extensive facial and orbital fractures are consistent with Le Fort type III fractures.

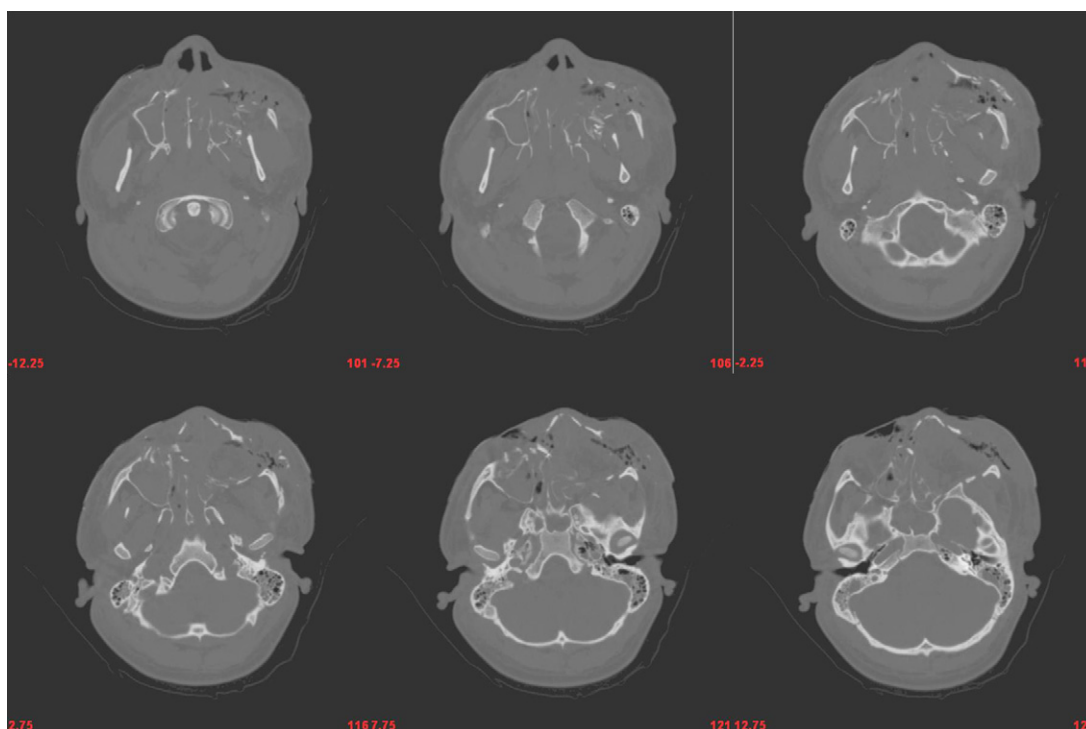


Fig. 17 CT series of a midface injury.

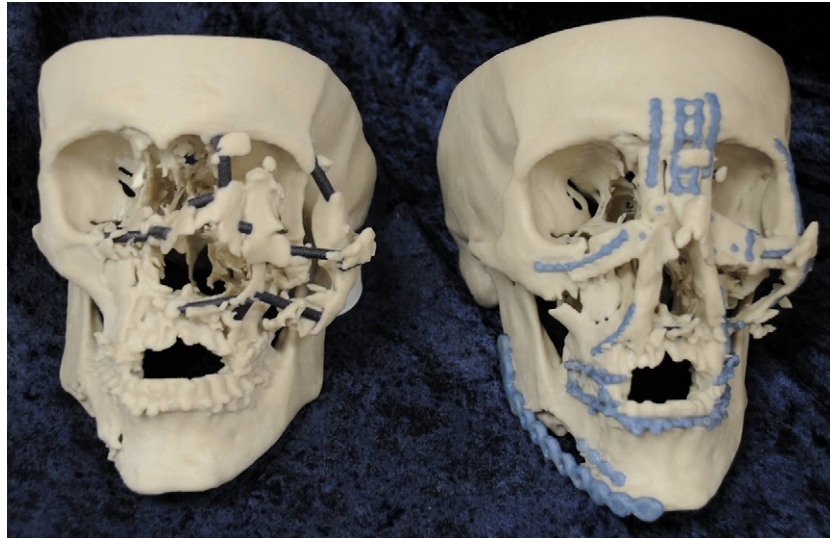


Fig. 18 Additive manufactured models from CT scans, before and after surgical reconstruction.

Surgeons use the preoperative model of the CT scan (**Fig. 18**) to generate a diagnosis and surgical plan. The relative locations of large bone fragments were maintained by incorporating black cylinders into the model. The color of the cylinders allows the surgical team to clearly identify bone. The post-operative model shows the patient with all fixations represented in light blue.

Custom cranial implant design and manufacturing

Digital files provide information to design and produce custom implants and devices. This technique is used frequently to produce custom cranial implants using the following processes:

- A CT image of the defect is generated as a computer model (**Fig. 19**).
- In the design process, mirror imaging was used to flip the unaffected skull; unaffected bone was then digitally cut and sculpted to produce an implant that perfectly fits the contours of the deficiency.
- Small fixation plates were then modeled and incorporated to the implant for attachments to the skull (**Fig. 20**).
- Model of the skull was manufactured on a stereolithography apparatus and the titanium implant was

manufactured on an electron beam-melting (EBM) machine (**Fig. 21**).

In a similar fashion, PMMA (polymethyl methacrylate) implants (**Fig. 22**) can be made by manufacturing an implant prototype on the stereolithography machine and then using conventional techniques to mold and manufacture the implant⁶ (**Fig. 23**).

Frontal implants, especially when involving the facial bones, prove to be a more complicated implant to design (**Fig. 24**). Preinjury pictures of the patient to obtain and create a properly contoured implant for the forehead are often helpful.

Severe maxillofacial injury

In severe facial trauma cases, facial bones can be modeled to show the full extent of the injury (**Fig. 25**). Using digital design techniques, a virtual reconstruction can be developed by the reconstruction team.

- Bony sections, such as the mandible's rami, can then be realigned to a "close-to-normal" position by using a mandible similar in size from another individual (**Fig. 26**).

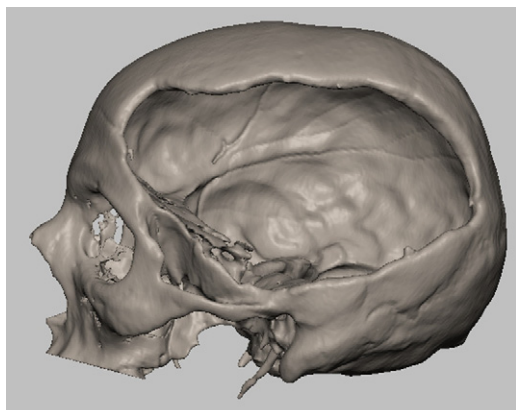


Fig. 19 Reconstruction of a cranial defect.

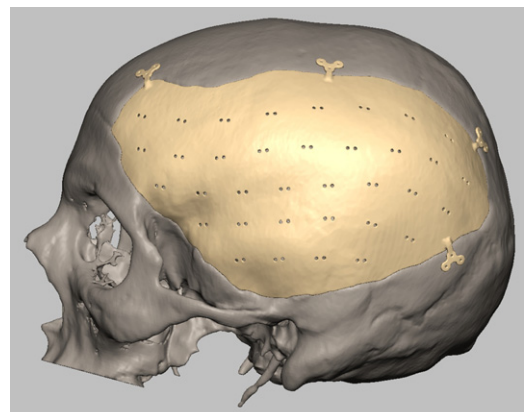


Fig. 20 Reconstructed cranial implant design.

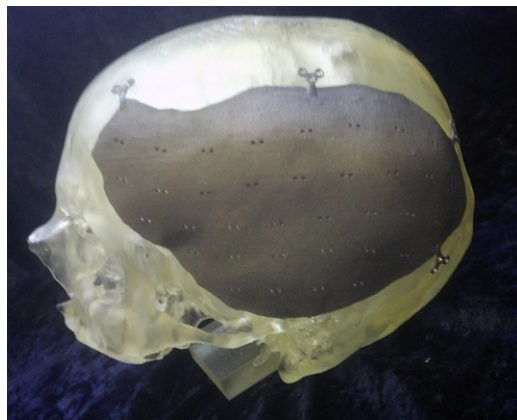


Fig. 21 Fabricated titanium cranial implant.

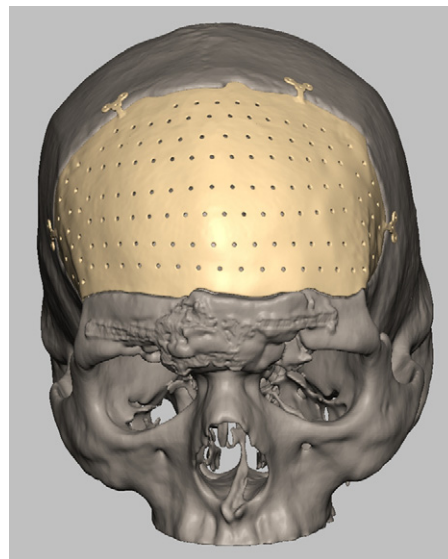


Fig. 24 Reconstructed frontal bone implant.



Fig. 22 PMMA implants for a frontal bone defect.

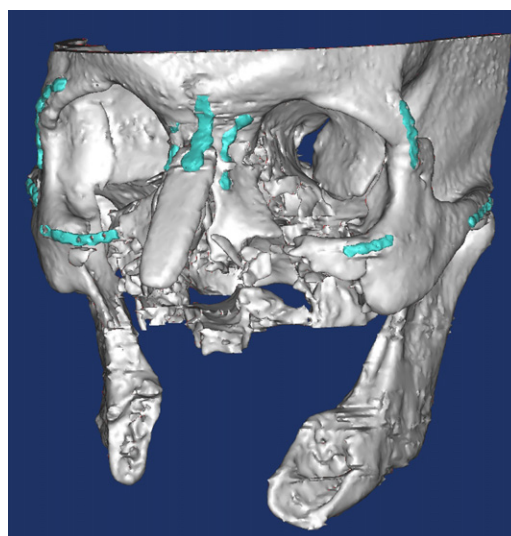


Fig. 25 Model of a maxillary/mandibular defect.

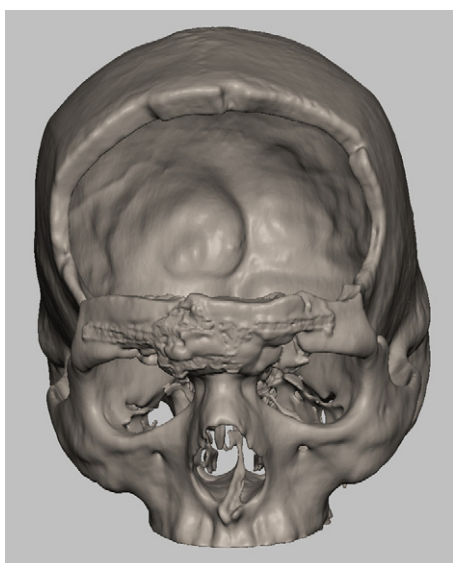


Fig. 23 Reconstruction of a frontal bone defect.

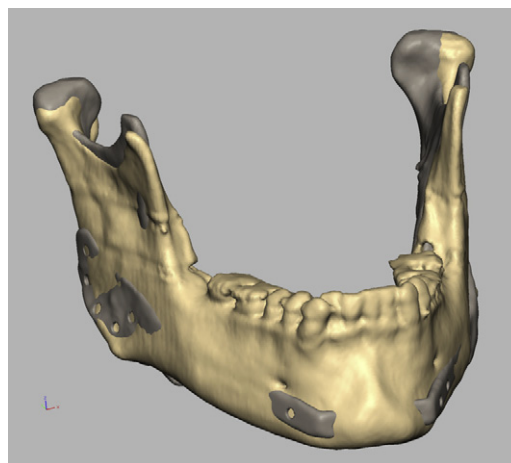


Fig. 26 Virtual realignment of the ramus sections from a similar mandible.

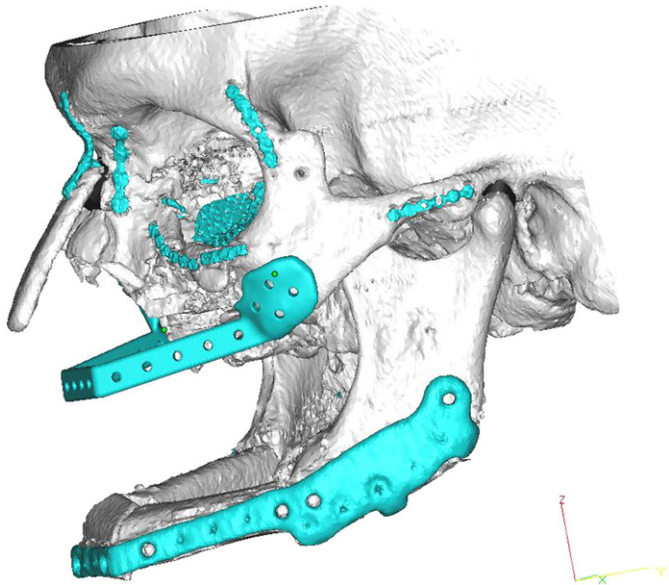


Fig. 27 Rendering of fixation for bone grafts of the maxilla/mandible.

- Once in their proper position, custom surgical fixation bars can be designed (Fig. 27).
- Digital fixation files can be milled using computer-aided design/computer-aided manufacturing or built directly with EBM or a laser sintering machine.
- If time does not allow for the use of custom fixation, the models can help to determine the size and prebend standard fixation plates before they enter the operating room.

Maxillofacial prosthetics

In cases in which facial trauma leads to the loss of soft tissue, facial prosthetics need to be created. CBCT, CT, and 3D camera systems all provide the information needed to virtually recreate a missing facial feature. Mold fabrication or direct fabrication of a custom facial prosthesis can be produced.^{7,8} As an example, using facial geometry obtained from a 3D camera system, a mirror image and alignment of digital ear reconstructed (Fig. 28). A mold can be designed around the ear, printed using additive manufacturing techniques, and silicone can be placed by the technician (Figs. 29 and 30). In the case of features such as the nose, where there are no features to

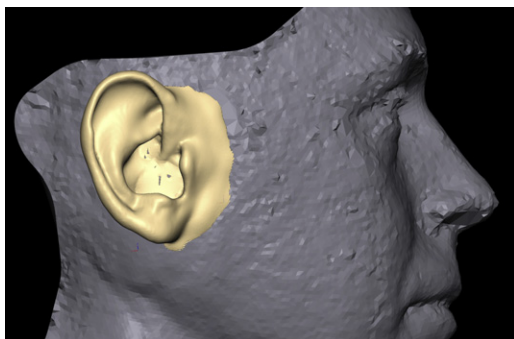


Fig. 28 Rendering of a mirrored ear to the defect side.

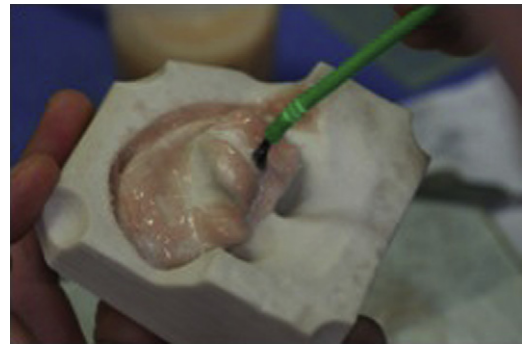


Fig. 29 Technician applying silicone to the ear mold.

mirror, a nose can be obtained from another individual and added digitally to the patient's anatomy to create the "ideal geometry" (Fig. 31).

Similar techniques using 3D photographic systems can be used to replace conventional impression techniques to produce casts for facial prostheses.^{9,10}

Custom plastic surgery guides

Digital images and software allow for virtual manipulation of the images as a 3D object which is ideal for many plastic procedures. Presurgical guides and cut patterns can be produced, minimizing operating time and increasing successful reconstructions.¹¹

- An ideal nose is selected from another individual, positioned, and then scaled to fit the patient's face. The nose is then adapted further to achieve smooth transitions to surrounding facial tissue. Preinjury pictures can provide addition information, such as width and depth, to the surgeon/engineer, thus making it easier to create the desired "ideal" nose (Fig. 32).
- Nose templates are digitally offset from the face and digitally cut to the desired shape. After verification and minor modifications, these guides are rapid prototyped and molded. Molds are then used to manufacture guides in PMMA. It has proved beneficial in the operating room to have both the entire nose and half-nose guides of the patient's "ideal" nose (Fig. 33).
- The ideal nose is covered in foil to achieve a grafting template. The "half nose" rhinoplasty guide (shown lying next to the face in Fig. 34), is made from PMMA. The transparency of the PMMA is an added benefit to the surgical procedure.

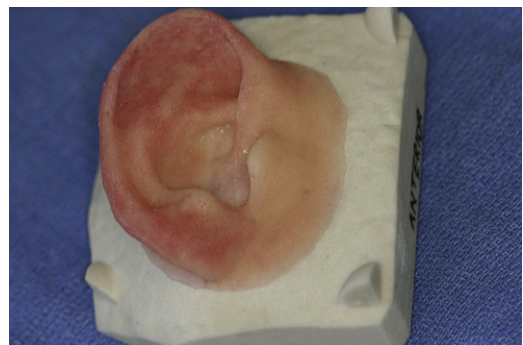


Fig. 30 Prosthetic ear on the mold.

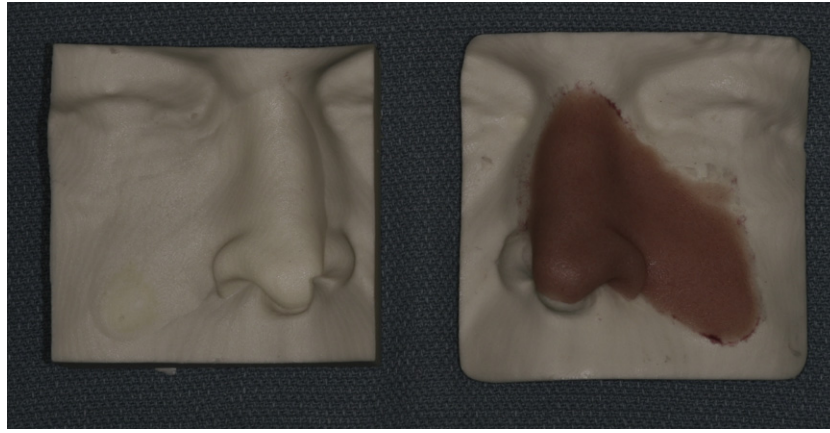


Fig. 31 Mold technique used for a nose.

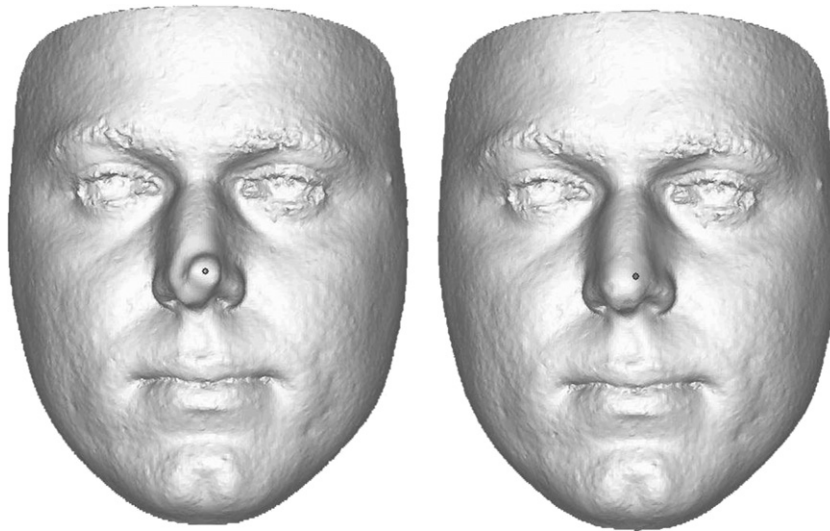


Fig. 32 Virtual rendering of pre and post virtual rhinoplasty.

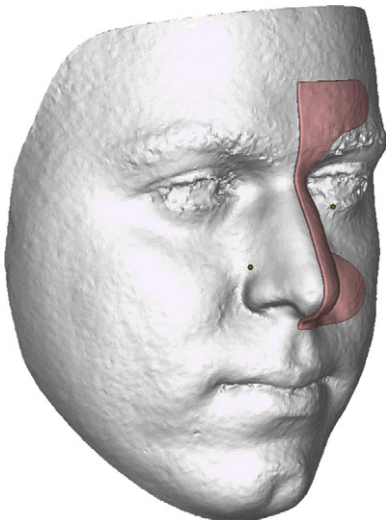


Fig. 33 Virtual design of soft tissue index for rhinoplasty.



Fig. 34 Fabricated guides for rhinoplasty, surgical foil is used to develop pattern for the soft tissue graft.

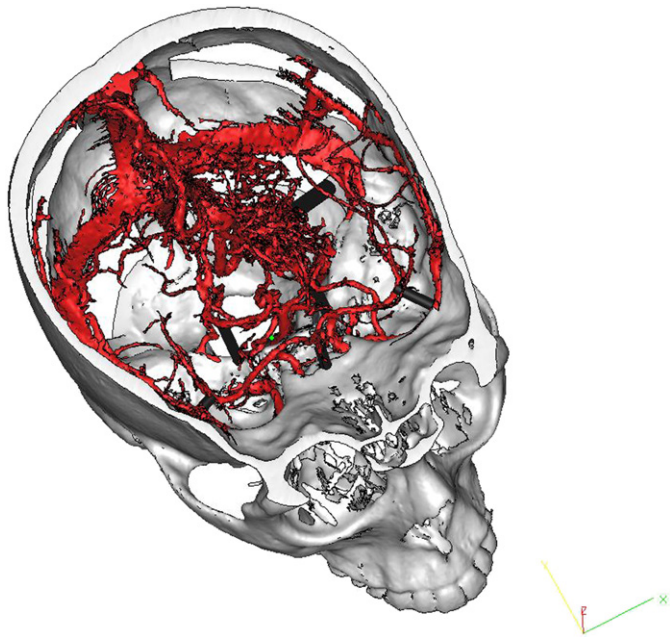


Fig. 35 Cranial model design for a vascular model.

Vascular modeling

Some trauma cases can lead to formations of aneurysms or arterial venous malformations. The ability to use isolate contrast within CT scans provides the opportunity to be able to segment out vessels. 3D models of the vessels can be developed and physical models can produce a "3D Road Map" that can be used to determine a surgical plan. These types of models can help determine the need for embolization, coiling, and clipping (Fig. 35).

Digital and 3D models can show fixations, arteries, veins, and monitors. Custom windows are added in the bone for neurosurgeons to achieve the best view to aid in their diagnosis and surgical plan (Fig. 36).

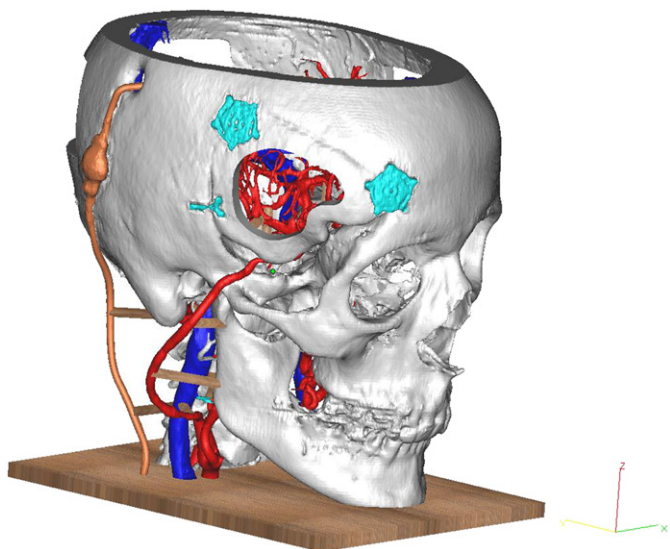


Fig. 36 Vascular model, note the access windows.

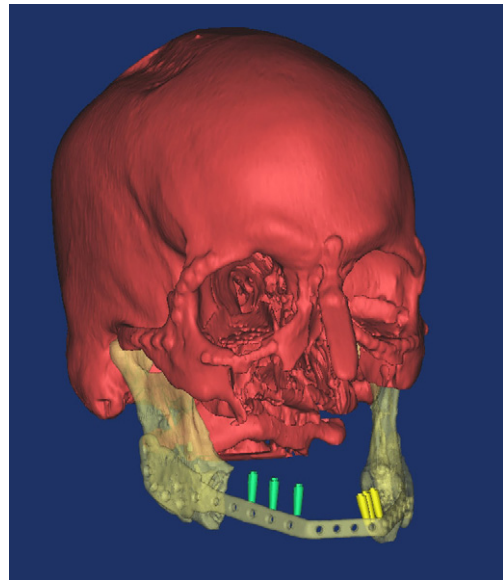


Fig. 37 Virtual placement of dental implants to accommodate the restorative plan in the presurgical computer model.

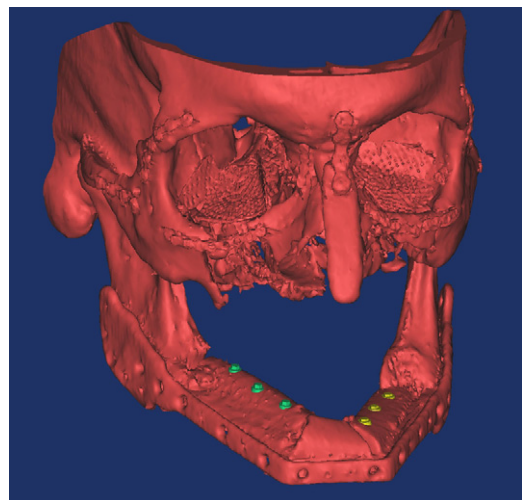


Fig. 38 Implant platforms on the grafted bone from the pre-surgical plan.

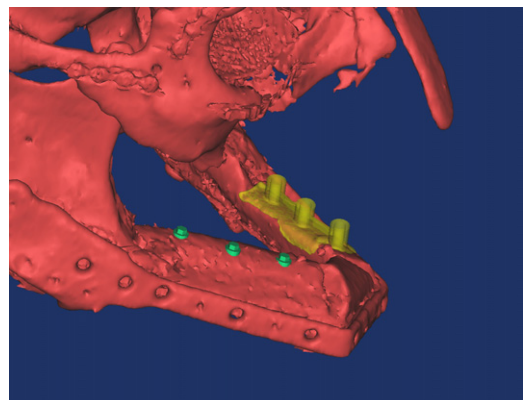


Fig. 39 Bony contoured surgical guide for dental implant fabrication.

Implant guides

In addition to visualization of the hard and soft structures that the CBCT and CT scans can provide, they have become more commonly used to predict the proper placement of dental implants. In more severe reconstruction cases, implants with differing properties, such as angulated platforms, can be placed in the proposed sites at the time of the presurgical bony restorations (Fig. 37). The surfaces can be calculated to the bone levels from the grafted bone (Fig. 38), and a guide for the placement can be designed and fabricated for placement to accommodate implants with both angulated and nonangulated implants (Fig. 39).¹²

References

1. Petrik V, Apok V, Britton JA, et al. Godfrey Hounsfield and the dawn of computed tomography. *Neurosurgery* 2006;58(4):780–7.
2. White SC, Pharoah MJ. *Oral radiology principles and interpretation*. Sixth edition. St Louis, MO: Mosby Elsevier; 2009. p 210.
3. Ludlow JB, Ivanovic M. Comparative dosimetry of dental CBCT devices and 64-slice CT for oral and maxillofacial radiology. *OOOOE* 2008;106(1):106–14.
4. Silva IM, Freitas DQ, Ambrosano GM, et al. Bone density: comparative evaluation of Hounsfield units in multislice and cone-beam computed tomography. *Braz Oral Res* 2012;26(6):550–6.
5. Chua CK, Leong KF, Lim CS. *Rapid prototyping: principles and applications*. Singapore: World Scientific; 2003.
6. Gronet PM, Waskewicz GA, Richardson C. Preformed acrylic cranial implants using fused deposition modeling: a clinical report. *J Prosthet Dent* 2003;90(5):429–33.
7. Liacouras P, Garnes J, Roman R, et al. Auricular prosthetic design and manufacturing using computed tomography, 3D photographic imaging and rapid prototyping. *J Prosthet Dent* 2011;105:78–82.
8. Ciocca L, Mingucci R, Gassino G, et al. CAD/CAM ear model and virtual construction of the mold. *J Prosthet Dent* 2007;98:339–43.
9. Sabol J, Grant G. Digital image capture and rapid prototypeing of the maxillary defect. *J Pros* 2011;20(4):310–4.
10. Ciocca L, Scotti R. CAD-CAM generated ear cast by means of a laser scanner and rapid prototyping machine. *J Prosthet Dent* 2004;92: 591–5.
11. Lindsay RW, Herberg M, Liacouras P. The use of three-dimensional digital technology and additive manufacturing to create templates for soft-tissue reconstruction. *Plast Reconstr Surg* 2012;130(4): 629e–31e.
12. Vercruyssen M, Jacobs R, Van Assche N, et al. The use of CT scan based planning for oral rehabilitation by means of implants and its transfer to the surgical field: a critical review on accuracy. *J Oral Rehabil* 2008;35(6):454–74.

Wound Management and Nutrition for Optimal Wound Healing

Steven V. Dryden, DDS ^{a,*}, William G. Shoemaker, DDS ^a, Jae H. Kim, DDS ^b

KEYWORDS

- Wound healing • Wound management • Nutrition • Vitamin • Macronutrients • Micronutrients • Wound care
- Wound dressing

KEY POINTS

- Wound healing occurs over 4 phases: (1) hemostasis; (2) inflammation; (3) proliferation; (4) remodeling.
- Macronutrients (proteins/amino acids, carbohydrates, and essential fatty acids) provide building blocks and energy for tissue growth, cell renewal, and repair after injury.
- Micronutrients (vitamins and minerals) enhance cellular proliferation and maintenance.
- Wound healing impediments include local and systemic factors.
- The goal of wound care is to optimize the environment through removal of necrotic tissue, foreign debris, bacterial load, and limit the amount of dead space.

Wound healing

Introduction

To obtain appropriate wound care and nutrition for optimal wound healing, it is important to understand the fundamentals of wound healing. Traditionally there are 4 phases (hemostasis, inflammation, proliferation, and remodeling) without distinct separation between the phases (Fig. 1). The complex interaction and timing of wound healing are critical to understand so as to obtain the best outcome for injured patients. Through a variety of chemical mediators (ie, cytokines, chemokines, growth factors, and inhibitors), the transition from wounding to healing takes place (Table 1).

Phases of wound healing

Hemostatic phase

Following tissue injury, the healing process begins with hemostasis as the body's mechanism to limit hemorrhage. Wounded cell membranes release vasoconstrictors, thromboxane A₂ and prostaglandin 2 α , promoting clot formation and

hemostasis (Fig. 2). As blood vessels constrict, circulating platelets become activated by the exposed collagen and release chemical mediators, promoting further platelet aggregation and activation. The exposed collagen also triggers the clotting cascade to form a fibrin matrix, which serves as the scaffold for the platelet plug and other invading cellular responders (ie, leukocytes, endothelial cells, fibroblasts). The platelet plug and adhesive proteins (fibronectin, vitronectin, and thrombospondin) form a provisional matrix, or fibrin clot. The provisional matrix is crucial for early hemostasis and prevents bleeding hours to days after injury.

Inflammatory phase

Following initial hemostasis, the injured cell membranes release chemical mediators, leading to the inflammatory phase lasting for 4 to 6 days. The inflammatory mediators (ie, prostaglandins) create vasodilation of proximal vessels, allowing for increased cellular response. Cardinal signs of the inflammatory process include erythema, heat, edema, and pain, and should resolve 48 to 72 hours after the wound has occurred. Platelet-derived growth factor and transforming growth factor- β , released by the platelets, are chemotactic for circulating neutrophils (PMNs) and monocytes, triggering them to enter the wound. Interleukin-1, tumor necrosis factor- α , and platelet factor 4 are additional chemotactic signals for PMNs. The endothelial cells adjacent to the wound are activated and form a molecule adhesion with the PMNs. The margination of PMNs along the vessel wall leads to diapedesis, the process of moving through the vascular wall into the wound bed (Fig. 3). PMNs clear the wounded site of bacterial and necrotic debris by releasing proteolytic enzymes. 48 to 96 hours, later circling monocytes are attracted and transformed into macrophages in the area. The macrophages release further cytokines and enzymes for wound healing. The cellular responders during the inflammatory phase promote early granulation tissue formation and tissue debridement, setting the stage for the proliferative phase.

Disclaimer: The views expressed in this article are those of the author and do not necessarily reflect the official policy or position of the Department of the Army, Department of the Navy, nor the US Government.

^a Division of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, MD 20889, USA

^b Oral and Maxillofacial Surgery Residency Program, National Capital Consortium, Division of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, MD 20889, USA

* Corresponding author.

E-mail address: steven.dryden@us.army.mil

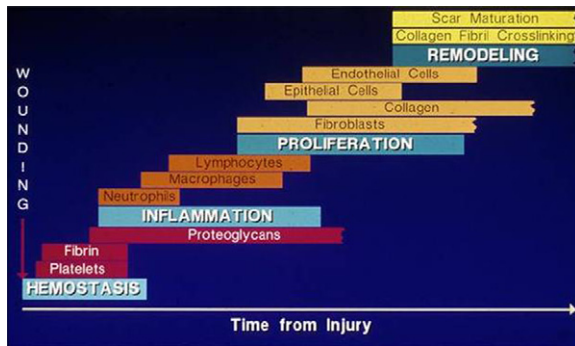


Fig. 1 Progression through the 4 major phases of wound healing: hemostasis, inflammation, proliferation, and remodeling. (From Cohen IK, Diegelmann RF, Lindblad WJ, editors. Wound healing: biochemical and clinical aspects. Philadelphia: WB Saunders; 1993; with permission.)

Proliferation phase

The transition from the inflammatory phase to proliferation occurs on about day 4, and progresses through day 14 (Fig. 4). Primary changes during the proliferation phase include epithelialization, angiogenesis, and granulation.

Epithelialization occurs either at an intact basement membrane or at the wound margin. It is stimulated by epidermal growth factor and transforming growth factor- α , products of activated platelets and macrophages. Fibroblasts synthesize and release keratinocyte growth factors and

interleukin-6, which activate keratinocytes to migrate over the wound, creating an early barrier.

Angiogenesis, stimulated by tumor necrosis factor- α , is the formation of new capillaries within the wound bed. The new blood vessels promote blood flow to manage the increased metabolic activity within the wound. Local factors leading to the increase in angiogenesis include low oxygen tension, low pH, and high lactate levels. As a result of angiogenesis, there is increased oxygen availability and immune-mediated cell access to cellular debris and bacterial contaminants for clearance.

Granulation tissue is a dense network of newly formed capillaries and blood vessels, fibroblasts and macrophages, and randomly deposited collagen fibers. The granulation tissue forms as fibroblasts migrate into the wound site from surrounding areas, become activated, synthesize collagen, and proliferate (Fig. 4). The capillaries carry nutrients and oxygen to support the elevated metabolic rate of cellular migration, division, and protein synthesis.

Remodeling phase

From about day 8 through about 1 year, the wound continues to transform and undergo maturation and remodeling (Fig. 5). Collagen synthesis continues for 4 to 5 weeks following wound formation. Initial collagen is thin and oriented parallel to skin. Over time, the thin collagen is absorbed and thicker collagen is deposited along the skin tension lines, thereby increasing tensile strength. As the remodeling progresses, collagen synthesis and degradation occur to re-create the tissue before injury; however, wound strength never reaches 100%. In fact, at 1 week,

Table 1 Summary of inflammatory cytokines

Cytokine	Cell of Origin	Function
EGF	Platelets, macrophages	Mitogenic for keratinocytes and fibroblasts, stimulates keratinocyte migration
FGF	Macrophages, mast cells, T lymphocytes, endothelial cells	Chemotactic and mitogenic for fibroblasts and keratinocytes, stimulates angiogenesis
IFNs (α , β , and γ)	Lymphocytes, fibroblasts	Activate macrophages, inhibit fibroblast proliferation
ILs (1, 2, 6, and 8)	Macrophages, mast cells, keratinocytes, lymphocytes	IL-1: induces fever and adrenocorticotrophic hormone release; enhances TNF- α and IFN- γ , activates granulocytes and endothelial cells; and stimulates hematopoiesis IL-2: activates macrophages, T cells, natural killer cells, and lymphokine-activated killer cells; stimulates differentiation of activated B cells; stimulates proliferation of activated B and T cells; and induces fever IL-6: induces fever and enhances release of acute-phase reactants by the liver IL-8: enhances neutrophil adherence, chemotaxis, and granule release
KGF	Fibroblasts	Stimulates keratinocyte migration, differentiation, and proliferation
PDGF	Platelets, macrophages, endothelial cells	Cell chemotaxis, mitogenic for fibroblasts, stimulates angiogenesis, stimulates wound contraction
TGF- α	Macrophages, T lymphocytes, keratinocytes	Mitogenic for keratinocytes and fibroblasts, stimulates keratinocyte migration
TGF- β	Platelets, T lymphocytes, macrophages, endothelial cells, keratinocytes	Cell chemotaxis stimulates angiogenesis and fibroplasia
Thromboxane A2	Destroyed wound cells	Potent vasoconstrictor
TNF	Macrophages, mast cells, T lymphocytes	Activates macrophages, mitogenic for fibroblasts, stimulates angiogenesis

Abbreviations: EGF, epidermal growth factor; FGF, fibroblast growth factor; IFN, interferon; IL, interleukin; KGF, keratinocyte growth factor; PDGF, platelet-derived growth factor; TGF, transforming growth factor; TNF, tumor necrosis factor.

From Lawrence W, Diegelmann R. Growth factors in wound healing. Clin Dermatol 1994;12:157; and Broughton G 2nd, Janis JE, Attinger CE. The basic science of wound healing. Plast Reconstr Surg 2006;117(Suppl 7):12S–34S; with permission.

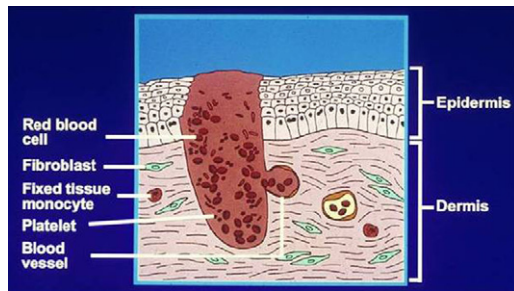


Fig. 2 Hemostatic phase. Following initial wounding, exposed collagen triggers platelet and fibrin clot formation to control hemorrhage. (From Greenfield LJ, editor. Surgery: scientific principles and practice. Philadelphia: J.B. Lippincott; 1993; with permission.)

3 weeks, and 3 months (and beyond), wound strength reaches 3%, 30%, and 80% respectively.

The balance between collagen deposition and degradation leads to scar formation. A fine linear scar is the equal balance between the 2 mechanisms. If more collagen is deposited, a hypertrophic scar will form. Adversely, if degradation is greater, wound dehiscence may occur.

Summary

The process of wound healing is a complex interaction composed of hemostasis, inflammation, proliferation, and remodeling. Understanding the phases of wound healing will give the surgeon a better understanding of how to optimize the healing process through proper nutrition and wound care management.

The role of nutrition in wound healing

"You are what you eat" is a saying that we are all very familiar with. From time immemorial, healers have known the value of good nutrition and the role it has had in the well-being of their patients. Some 2300 years ago, Hippocrates warned of underestimating the vital role that nutrition played in health and human disease. The healing process results from a complex series of events that involves the immune system working with many other physiologic systems. Digestion, absorption, protein synthesis, caloric needs, protein degradation, and hormonal control are all parameters that play a role in enabling the body to heal itself. As practicing surgeons, we are fortunate that the

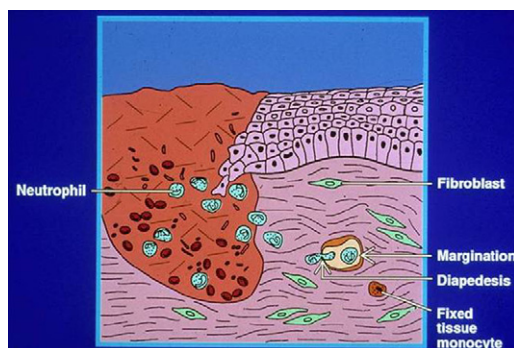


Fig. 3 Inflammatory phase. Platelet degranulation and inflammatory mediators lead to vasodilatation and ingress of inflammatory cells to debride the wound. (From Greenfield LJ, editor. Surgery: scientific principles and practice. Philadelphia: J.B. Lippincott; 1993; with permission.)

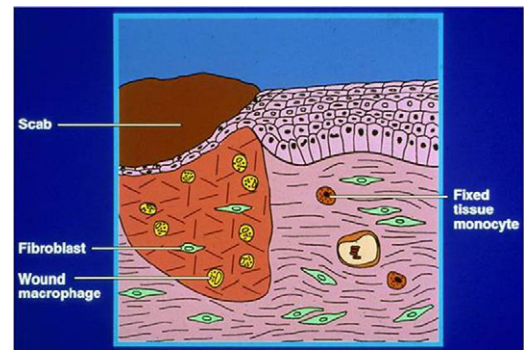


Fig. 4 Proliferative phase. Macrophages move into the wound bed, release cytokines, such as collagenase, to debride the wound; interleukins and tumor necrosis factor to stimulate fibroblasts, collagen formation, and promote angiogenesis; and transforming growth factor to stimulate keratinocyte activation. (From Greenfield LJ, editor. Surgery: scientific principles and practice. Philadelphia: J.B. Lippincott; 1993; with permission.)

vast majority of our patients are healthy and usually well-nourished and may be returned to a normal diet in a very short period of time. However, for those patient populations that may be at risk of malnutrition, such as the geriatric, diabetic, and those with impairments in nutrient bioavailability, the clinician must be ready to identify and treat these patients to ensure proper wound healing.

Nutrition screening

As with all new patients, a thorough history and a physical are conducted. These alone have been found to be 80% to 90% accurate in evaluating patient nutritional status. The addition of multiple or complex biochemical, immune, or anthropometric measurements does not increase greatly the accuracy of nutritional assessment. In fact, some studies indicate that anthropomorphic measurements, such as body mass index and weight loss, are less sensitive markers of malnutrition. Although low body mass index values and recent weight loss are associated with hypoalbuminemia, such measures can miss more than half of patients with protein deficiency. As such, anthropomorphic measures likely are insufficient for identifying patients who might benefit from a laboratory nutritional assessment. However, the clinician has at his or her disposal

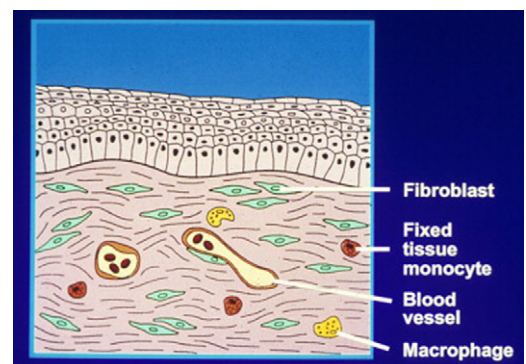


Fig. 5 Maturation phase. Early disorganized collagen is replaced by organized collagen to more closely resemble surrounding tissue. (From Greenfield LJ, editor. Surgery: scientific principles and practice. Philadelphia: J.B. Lippincott; 1993; with permission.)

several nutrition screening tools that can be used rather easily. Validated nutrition screening tools include the Mini-Nutritional Assessment-Short Form (MNA-SF), the Malnutrition Universal Screening Tool (MUST), and the Malnutrition Screening Tool (MST). The MUST nutritional risk screen identifies adults who are underweight and at risk of malnutrition. It has been validated in acute care, long-term care, and in the community. The MST screening tool is valid and reliable for identifying nutrition problems in acute care and ambulatory care. The MNA-SF was revised to a 6-item screening tool and revalidated as a stand-alone screening tool. The tool has 3 cutoff points, allowing clinicians to quickly identify those who are malnourished. The maximum score is 14. Scores of 12 to 14 indicate well nourished, scores of 8 to 11 indicate nutrition risk, and scores of 0 to 7 indicate that the individual is malnourished. The MNA-SF has been validated to identify malnutrition in older adults, age 65 and older, residing in the community or institutional settings. It has an 80% sensitivity specificity and 97% positive predictive value, according to clinical studies.

Currently, there is no standard regimen for testing or monitoring nutritional deficiencies in wound patients. Some researchers suggest that systematic laboratory nutritional assessments and C-reactive protein levels may be appropriate. Moreover, there is a dearth of information for testing local wound nutritional deficiencies outside the research setting.

Macronutrients

Protein and amino acids

Proteins provide the main building blocks for tissue growth, cell renewal, and repair after injury. They significantly affect multiple phases of wound healing (hemostasis, inflammation and granulation tissue formation, cell proliferation, tissue reorganization, and normalization) by their roles in RNA and DNA synthesis, collagen and elastic tissue formation, nutrition of the immune system, epidermal growth, and keratinization. With prolonged protein malnutrition, skin becomes thinner and wrinkled and immunity wanes. Diabetic patients with protein malnutrition are at higher risk for amputations.

Dietary proteins that provide all 9 of the essential amino acids are considered complete proteins. Food sources of complete protein include meat, poultry, fish, eggs, milk products, and soybeans. The body needs an adequate supply of essential amino acids, enough nitrogen and energy for the synthesis of the 11 other amino acids. Legumes, grains, and vegetables provide incomplete proteins.

Certain nonessential amino acids become conditionally essential during periods of trauma, such as thermal injury, sepsis, or pressure ulcers. 1-Arginine is 32% nitrogen and in some studies has been shown to increase concentrations of hydroxyproline, which is an indicator of collagen deposition and protein in the wound site. Glutamine has been shown to be used by inflammatory cells within the wound for proliferation and as a source of energy.

Carbohydrates

With regard to nutrition, the body's main concern is for adequate energy provided from carbohydrate, protein, and fat. When the total amount of calories consumed is too low, protein from both the diet and the individual's muscle stores will be used as an energy source, thus increasing the caloric requirements needed to promote anabolism and reverse catabolism.

Carbohydrates provide energy and prevent gluconeogenesis when the body is forced to convert protein stores for energy

use. An inadequate supply of carbohydrates can lead to muscle wasting, loss of subcutaneous tissue, and poor wound healing. Grains, fruits, and vegetables with complex carbohydrates are the preferred sources.

Lipids and essential fatty acids

The most concentrated source of energy comes from fats and triglycerides, which provide energy for proliferation and are building blocks for epidermal and dermal tissues. They are important for cell membrane synthesis, epidermal phospholipids, inflammatory reactions, and intracellular matrix synthesis.

Micronutrients

Vitamins

Water-soluble vitamins B and C are absorbed into the bloodstream and are excreted if blood concentrations are too high. Although foods do not deliver toxic doses of water-soluble vitamins, large amounts in supplements can reach toxic levels.

Vitamin B complex consists of 8 water-soluble vitamins found in meat, dairy, vegetables, fish, and cereals. Vitamin B complex helps to promote cell proliferation and maintain healthy skin and muscle tone, increase metabolic rate, and enhance immune and nervous system function. Deficiencies in vitamin B can impair wound healing and are associated with several disorders, many of which have skin manifestations. In particular, thiamine is associated with decreased wound healing and breaking strength.

Vitamin C enhances activation of leukocytes and macrophages in the wound bed and is essential for collagen synthesis. A deficiency of vitamin C prolongs the healing time and contributes to reduced resistance to infection. To date, there is no clinical evidence that wound healing is improved by providing mega-doses of vitamin C above the Dietary Reference Intake (DRI of 70–90 mg/d). Good sources of ascorbic acid are citrus fruits, strawberries, tomatoes, potatoes, broccoli, mangoes, and green peppers.

Fat-soluble vitamins A, D, E, and K dissolve in fat and are transported in the body attached to lipids. Unlike water-soluble vitamins, they are stored in the liver and fatty tissue until blood concentrations decline and the body retrieves them from storage.

Vitamin A is responsible for epithelium maintenance and it also stimulates cellular differentiation into fibroblasts and collagen formation. It has also been shown to reverse the anti-inflammatory effects of corticosteroids on wound healing. The administration of vitamin A, topically or systemically, also can correct the impaired wound healing of patients on long-term steroid therapy. This increase of the inflammatory response is thought to occur by an enhanced lysosomal membrane lability, increased macrophage influx and activation, and stimulation of collagen synthesis. These mechanisms still are not well understood, but it is clear vitamin A plays an important role in wound healing. Vitamin A deficiency, which is uncommon, may result in delayed wound healing and increased susceptibility to infection. Good sources of vitamin A are carrots, sweet potatoes, apricots, spinach, and broccoli.

Vitamin D, a fat-soluble vitamin, is involved in calcium uptake and metabolism by inhibiting secretion of calcitonin and parathyroid hormone. Vitamin D is readily obtained from sunlight, fatty fish, whole eggs, beef liver, mushrooms, and fortified foods. Deficiency in vitamin D leads to rickets in children and osteomalacia and osteoporosis in adults. The role of vitamin D in wound healing is unclear.

Vitamin E, another fat-soluble vitamin, serves as an antioxidant role interacting with selenium-dependent glutathione oxidase to inhibit degradation of cell membrane fatty acids. Low levels of vitamin E have been reported in chronic wound patients. In chronic wounds, free radical formation is enhanced because of the inflammatory cascade caused by ischemia, necrotic tissue, and microbial flora. Vitamin E is found in asparagus, avocados, eggs, nuts, and spinach. Supplementation remains controversial. Some reports indicate that vitamin E may impair collagen synthesis and wound healing in animals, whereas other investigators report enhanced healing in irradiated rat skin and patients with post-thrombotic leg ulcers.

Vitamin K, also a fat-soluble vitamin, is present in leafy green vegetables, parsley, kiwi, meat, eggs, and dairy. Vitamin K is needed for posttranslational modification of certain proteins that are mainly required for coagulation and bone metabolism. Deficiency can result in hemorrhage, impaired wound repair, and infection.

Minerals

Iron is important in hemoglobin formation and oxygen transport, uptake, and metabolism of free radicals, and hydroxylation of collagen precursors. Iron deficiency interferes with healing through tissue hypoxia and decreased bactericidal ability by leukocytes.

Zinc is a cofactor for at least 70 major enzyme systems important in wound healing, including DNA and RNA polymerases, proteases, and carbonic anhydrase. It also liberates vitamin A from storage in the liver and assists in immune function. Many studies have reported significantly lower zinc levels in chronic wound patients compared with presumably healthy controls. Because zinc deficiency impairs wound healing, zinc repletion may increase healing rates; however, there is no strong clinical evidence that oral zinc sulfate aids healing of arterial and venous ulcers. Topical zinc acts as a mild antiseptic and anti-inflammatory agent in wound care, whereas one study demonstrated that 1% zinc oxide cream increased mitosis and reepithelialization rates.

Water

Water is critical for optimal healing. Hydration promotes cell proliferation and migration along chemotactic gradients created by metal ions, cytokines, and growth factors. Dehydration leads to epidermal hardening and dermal necrosis that delays wound healing and adds to patient discomfort.

Clinical implications

The wound-healing phase is extremely energy demanding. There is a strong increase in cell proliferation, protein synthesis, and enzyme activity during the healing process that requires energy and building substrates. Normally, these substrates are released from body energy stores and protein reserves; however, undernourished subjects need increased food intake or supplements with high energy and protein density. In addition, basic macronutrients, such as protein or amino acids, carbohydrate, fat and electrolytes, and micronutrients are necessary.

The daily energy requirement of a healthy person is 30 to 35 kcal/kg of body weight, depending on physical activity. In diseases, such as the usual multiple morbidities of a geriatric patient with coexisting wounds, energy intake should be increased to 35 to 40 kcal/kg per day. And although there are studies that support the use of specialized nutritional support in postoperative and wounded patients, this evidence has not

been borne out by clinical investigation. Also, the risk complications and increased cost of these specialized nutritional support elements need to be considered as well. Indeed, nutrition and nutritional supplementation in wound care is not yet standard of care and remains controversial.

Preoperative nutritional support is generally recommended for patients with moderate (10%–20% weight loss; serum albumin <3.2 g/dL to >2.5 g/dL) to severe malnutrition (>20% weight loss; serum albumin <2.5 g/dL) and who can tolerate waiting at least 7 days for an elective operation. If intestinal function is maintained in a patient, enteral nutritional support is generally preferred, as it is associated with the maintenance of gut mucosal barrier function, the decreased activation of gut-associated lymphoid tissue, and lower costs of administration than parenteral nutrition. Total parenteral nutrition is reserved for patients with ineffective gastrointestinal function, not compromised oral function.

Serum protein markers are the best way to assess the adequacy of nutritional supplementation, as conventional methods, such as daily weight, may not be accurate in critically ill patients. Although albumin is commonly used as a preoperative marker of nutrition, its half-life of 18 to 21 days precludes its use as an effective daily indicator of improvements in nutritional status. Prealbumin (half-life 3–5 days) and transferrin (half-life 7–10 days) should be monitored weekly in patients receiving enteral or parenteral nutritional support.

In general, it is important to counsel undernourished patients about ways to improve their diets. Providing nutritional supplements in addition to regular food intake seems a logical means of replenishing nutrients and supplying extra nutrients for increased tissue resistance and wound repair.

Summary

Nutrition and its role in wound healing has been the subject of intense study and experimentation. New research is pointing to exciting nutritional interventions that will advance not only our understanding, but more importantly, better outcomes for our patients. Without a doubt, poor nutrition leads to poor outcomes, whereas the reverse has a positive effect on wound healing. The clinician must recognize those patients who have poor nutrition or are at risk and address the patient's needs accordingly to ensure successful wound healing and avoid wound failure.

Wound care management

To optimize the healing process, the provider must identify and remedy the cause of the underlying wound, provide localized wound care, including preparation of the wound bed and appropriate dressings, and provide support to ensure outcomes are met.

Identify and treat cause

The first step in wound care is to fully assess the patient and the wound by determining the mechanism of injury and the patient's health and nutritional status for optimal healing. Both local and systemic factors can lead to impaired wound healing (Table 2). Local factors directly alter the characteristics of the wound itself; whereas, systemic factors are the complete disease state affecting one's ability to heal. Many of these factors are related, and the systemic factors persist

Table 2 Factors affecting wound healing

Local		Systemic
Ischemia	Age	Medication
Infection	Diabetes mellitus	Alcoholism
Foreign body	Hypothyroidism	Smoking
Edema	Stress	Nutrition
	Obesity	Corticosteroids

through the local effects disturbing wound healing, potentially turning the acute wound into a chronic one. The classification of acute versus chronic wounds is determined by the orderly and timely manner for which the healing process takes place to restore form and function. Chronic wounds do not undergo the normal progression of hemostasis, inflammation, proliferation, and remodeling, and instead remain in a chronic inflammatory state.

Wound care

The etiology of wounding and suspicion for contamination will help dictate the steps of managing the wound. A contaminated open wound with a high bacterial load ($>10^5$ organisms/gram of tissue or with β -hemolytic streptococcal species) and/or foreign debris must be kept open until the environment changes. Removal of necrotic tissue, foreign debris, bacterial contamination, and limiting dead space through appropriate dressings will help optimize the wound bed. A wound with dead space (depth, tunneling, or undermining) requires a wound filler to minimize accumulation of exudate and abscess formation. Covering a wound with an occlusive or semioclusive dressing, along with less frequent dressing changes will protect the wound from outside contaminant, trauma, and cold stress. Maintaining a normal wound bed temperature will prevent wound vasoconstriction and hypoxia and will decrease the risk of infection.

During normal wound healing, tissue defects progress through a series of coordinated molecular and cellular events, resulting either in regeneration or tissue repair. Primary intention, the least complicated wound repair, is the healing of clean wounds without loss of tissue and uninfected surgical incisions approximated by sutures. Infected, contaminated wounds or wounds with poor vascular supply require a delayed healing process through secondary intention. The wound fills with granulation tissue, contracts, and reepithelializes. In comparison with healing by primary intention, this process takes longer and large amounts of granulation tissue are formed to fill the tissue defect, leading to a more extensive scar from a prolonged inflammatory phase. Tertiary closure (delayed primary closure) provides a superior cosmetic appearance following the closure of a contaminated wound. The wound is allowed to stay open to undergo repeated dressing changes to decrease the bioburden and thereby decrease the infection rate after delayed surgical closure.

Wound bed preparation

The wound bed must be prepared to promote wound health and appropriate healing. Wound treatment includes cleansing, wound debridement, and dressing. Warm physiologic solution is used to gently cleanse a wound to remove loose debris and lessen the amount of bioburden. Wound debridement provides removal of tissue and can be done surgically, autolytically, mechanically, and enzymatically. Surgical debridement allows

the surgeon to excise small to large quantities of necrotic tissue, thereby transforming the wound from a chronic to an acute process. Debridement via an autolytic process uses wound dressings and patient-produced proteolytic enzymes, separating the healthy granulation tissue from devitalized tissue. As the dressings are changed, the wound is irrigated and debrided to remove the created slough. Mechanical debridement is the nonselective removal of both vital and nonvital tissue via dressing changes (ie, wet to dry), pulse lavage, or aggressive irrigation. Enzymatic debridement occurs as enzymes are applied to the wound, creating a sloughing of tissues. Enzymes can be selective or nonselective for necrotic tissue. Examples include exogenous collagenases to facilitate debridement by degrading collagen and elastin and stimulating granulation tissue formation.

Wound dressings

Ideal wound dressings promote wound healing by maintaining a moist wound environment while absorbing excess exudate, provide mechanical protection, aid in debridement, are non-adherent to the wound, allow gaseous exchange but are impermeable to microorganisms, are easy to use, and are cost effective. Moist wound environments facilitate cellular growth and collagen proliferation. In contrast, dry wound tissue is prone to infection, scarring, delayed healing, and pain. Excessive moisture can impair the healing process and cause peri-wound maceration.

There are a multitude of wound care dressings available on the market (Table 3) and dressings may change through the various phases of healing (Fig. 6). Primary dressings are applied directly to the wound, whereas secondary dressings are used either as an adjunct to augment the therapeutic function of the primary dressing or used to secure the primary dressing.

Dressings are classified as open, semiopen or semioclusive. Open dressings include wet-to-dry dressings, such as moistened gauze placed directly in contact with the wound. The moistened gauze is applied to the wound bed, allowed to dry, removed, and replaced multiple times daily. This process can be very painful for the patient. The removal of the gauze is nonselective in debridement of the wound bed and often removes healthy granulation tissue. In addition, fibers from the gauze can become imbedded within the wound, creating a nidus for infection or foreign body reaction. As a result, wet-to-dry is no longer standard of care in wound therapy but can be useful in cellulitis, infection, insensate patients who need mechanical debridement, and short-term use. Alternatives to wet-to-dry dressings include hydrofibers or foams that have become the standard of care in wound management.

Semiopen dressings (contact layers) consist of fine mesh gauze impregnated with petroleum, paraffin wax, or other ointment. These dressings are intended for direct use on superficial wounds, skin tears, partial-thickness and full-thickness skin grafts, skin abrasions and lacerations, and second-degree burns. They conform well, are generally porous and allow fluid to be pulled through to the secondary dressing, and are nonadherent to moist wound beds, reducing pain during dressing changes. Additionally, they can remain in place when changing secondary dressing. Contact layers must be used in conjunction with a secondary dressing to absorb drainage. They cannot be used on wounds with tunneling, stage I pressure ulcers, or third-degree burns.

Semioclusive dressings (ie, film, hydrocolloid, hydrofiber, wound VAC) come in a wide variety of occlusive properties,

Table 3 Modern classes of dressings

Class	Description	Tissue Debridement	Infection	Moisture Balance	Indications/ Contraindications
1. Films/membranes	Semipermeable adhesive sheet; impermeable to water molecules and bacteria	+	—	—	Moisture vapor transmission rate varies from film to film. Should not be used on draining or infected wounds. ^a Create occlusive barrier against infection.
2. Nonadherent	Sheets of low adherence to tissue	—	—	—	Allow drainage to seep through pores to secondary dressings. Facilitate application of topical.
3. Hydrogels	Nonmedicated tulle Polymers with high water content	++	—/+	++	Should not be used on draining wounds. Solid sheets should not be used on infected wounds.
4. Hydrocolloids	Available in gels, solid sheets, or impregnated gauze May contain gelatin, sodium carboxymethylcellulose, polysaccharides, and/or pectin; sheet dressings are occlusive with polyurethane film outer layer	+++	—/+	++	Should be used with care on fragile skin. Should stay in place for several days. Should not be used on heavily draining or infected wounds. ^a Create occlusive barrier to protect the wound from outside contamination. Odor may accompany dressing change and should not be confused with infection.
5. Acrylics	Clear acrylic pad enclosed between 2 layers of transparent adhesive film	+++	—/+	++	Use on low-draining to moderately draining wounds in which dressing may stay in place for extended time. May observe wound without changing.
6. Calcium alginates	Sheets or fibrous ropes of calcium sodium alginate (seaweed derivative); have hemostatic capabilities	++	+	+++	Should not be used on dry wounds. Low tensile strength—avoid packing into narrow deep sinuses. Bioreabsorbable.
7. Composite dressings	Multilayered, combination dressings to increase absorbency and autolysis	+	—	+++	Use on wounds in which dressings may stay in place for several days. ^a
8. Foams	Nonadhesive or adhesive polyurethane foam; may have occlusive backing; sheets or cavity packing; some have fluid lock	—	—	+++	Use on moderately to heavily draining wounds. Occlusive foams should not be used on heavily draining or infected wounds. ^a
9. Charcoal	Contains odor-absorbing charcoal within product	—	—	+	Some charcoal products are inactivated by moisture. Ensure dressing edges are sealed
10. Hypertonic	Sheet, ribbon, or gel impregnated with sodium concentrate	+	+	++	Gauze ribbon should not be used on dry wounds. Maybe painful on sensitive tissue. Gel may be used on dry wounds.
11. Hydrophilic fibers	Sheet or packing strip of sodium carboxymethylcellulose; converts to a solid gel when activated by moisture (fluid lock)	+	—	+++	Best for moderate amount of exudates. Should not be used on dry wounds. Low tensile strength—avoid packing into the narrow deep sinus.
12. Antimicrobials	Silver, iodides, polyhexamethylene biguanide, honey aniline dyes with vehicle for delivery: sheets, gels, alginates, foams, or paste	+	+++	+	Broad spectrum against bacteria. Should not to be used on patients with known hypersensitivities to any product components.

(continued on next page)

Table 3 (continued)

Class	Description	Tissue Debridement	Infection	Moisture Balance	Indications/Contraindications
13. Other devices	Negative-pressure wound therapy applies localized negative pressure to the surface and margins of wound	—	+	+++	This negative pressure—distributing dressing actively removes fluid from wound and promotes wound edge approximation. Advanced skill required for patient selection for this therapy.
14. Biologics	Living human fibroblasts provided in sheets at ambient or frozen temperature; extracellular matrix Collagen-containing preparations; hyaluronic acid, platelet-derived growth factor	—	—	—	Should not be used on wounds with infection, sinus tracts, or excessive exudates. or on patients known to have hypersensitivity to any of the product components. Cultural issues related to source. Advanced skill required for patient selection for this therapy.

— No activity.

+ Minimal activity.

++ Moderate activity.

+++ Strong activity.

^a Use with caution if critical colonization is suspected.

Adapted from Canadian Association of Wound Care. Best Practice Recommendations for Wound Management: Putting Knowledge into Practice. A Seminar Series. 2005; *From* Sibbald RG, Goodman L, Woo KY, et al. Special consideration in wound bed preparation 2011: an update. *Adv Skin Wound Care* 2011;24(9):415–36; with permission.

absorptive capacities, conformability, and bacteriostatic activity.

Films

Polymer films are transparent sheets of synthetic self-adhesive dressing that are permeable to gases, such as water vapor and oxygen, but impermeable to larger molecules, including proteins and bacteria. Transparent film dressings (ie, Tegaderm [3M Corporation, St. Paul, MN]) are suitable for shallow partial-thickness wounds with minimal exudates. Alternatively, films are often used as secondary dressings.

Hydrocolloid and hydrofiber dressing

Hydrocolloid dressings typically consist of a gel or foam on a carrier of self-adhesive polyurethane film. They provide a moisture-balanced environment allowing clean wounds to granulate and necrotic wounds to debride autolytically. The colloid composition traps the exudates and bacteria and do not adhere to the moistened wound bed. The dressing change is a gentle, painless form of mechanical debridement. An additional advantage of hydrocolloids is the ability to use them for packing wounds. Disadvantages include malodor, the need for daily dressing changes, and allergic contact dermatitis with the adhesive contact layer.

Hydrocolloid dressings come in different thicknesses and can be used as a primary or secondary dressing. The dressing may be worn up to 7 days. It will not adhere to a moist wound bed, but will adhere to surrounding dry tissue. It decreases the risk of maceration to surrounding skin, lessens trauma to the wound, and reduces pain for the patient on removal. Mepilex Border Lite (Mölnlycke Health Care AB, Norcross, GA), which has thin Mepilex foam dressing with a self-adherent border, is ideal for a wound with irregular contours and dresses challenging areas, such as maxillofacial wounds.

Hydrofiber is indicated for moderate to high exudating wounds that are infected or at risk for infection/colonization.

Many hydrofibers are impregnated with silver (Ag), providing a bactericidal effect, particularly against gram-negative species, such as *Enterobacter* species, *Proteus* species, and *Escherichia coli*. The broad-spectrum antimicrobial effect of silver is also effective against gram-positive bacteria, with minimal chance for resistance. Hydrofibers are highly absorptive, are useful for deep cavity wounds, and do not adhere to a moist wound site. Hydrofibers turn to gel when wet, and often take on the appearance of the wound.

Wound VAC: the vacuum-assisted closure (negative-pressure therapy)

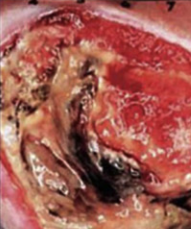
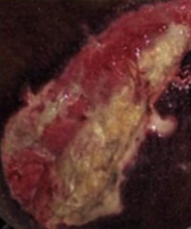
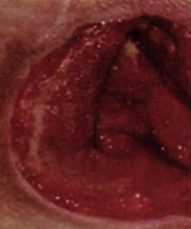
Vacuum-assisted closure (VAC) is also known as negative-pressure therapy because of the application of a controlled subatmospheric pressure to a wound covered with a foam dressing. The wound VAC system is useful for large avulsive defects that are difficult to close primarily (Fig. 7). The negative pressure removes interstitial fluid and edema to improve tissue oxygenation. It also removes inflammatory mediators that suppress the normal progression of wound healing. The wound VAC decreases bacterial counts to allow spontaneous healing and granulation tissue formation sooner than other methods. The negative pressure dressing is convenient to use and requires changing every 48 to 72 hours with minimal complications.

Wound care and patient support

For chronic wounds, it is important to develop a wound-care plan to provide long-term support, appropriate nutritional care, dressing instruction, and management. Many institutions have a wound-care team that may contain the following members:

- Physicians
- Wound-care nurses
- Institutional nurses

Walter Reed Bethesda Wound Dressing Selection Guide

Wound Appearance								
Description	Eschar* (Colors may vary)	Predominantly Slough (Infection may be present)	Granulating/ Mixed Wound Tissue	Fibrin (Appears yellow)	Granulating and/or Epithelializing	Skin Tear	Epithelializing	Healed Wounds, Skin at Risk or Closed Surgical Incisions
Exudate Level	 Moderate to None	 High  to Moderate			 Moderate  to Scant			 None
Depth	Unknown	Deep	Deep/Shallow	Deep/Shallow	Deep/Shallow	Shallow	Shallow	Closed
Treatment Objective	Debride*	Cleanse, Debride, Absorb, Fill Dead Space			Protect, Hydrate, Fill Dead Space			Protect
Suggested Products and Change Rates To the right are management options for each wound condition	Carrasyn® V Gel or Collagenase (needs Rx) (Daily) Cover choices: Alldress® or Mepilex® Border or Mepilex® Border Lite	Iodosorb® Gel (Every 2 days) or Melgisorb® (Up to 4 days) or Aquacel® Cover choices: Alldress® or Mepilex® or Mepilex® Border	<u>Deep</u> Iodosorb® Gel (Every 2 days) or Melgisorb® (Up to 4 days) or Aquacel® Cover choices: Alldress® or Mepilex® Border	<u>Moderate Exudate</u> Iodosorb® Gel (Every 2 days) or Melgisorb® (Up to 4 days) or Aquacel® Cover choices: Alldress® or Mepilex® Border	<u>Moderate Exudate</u> Melgisorb® (Up to 4 days) or Aquacel® Cover choices: Alldress® or Mepilex® Border	<u>Skin Tear Prevention</u> Tubifast® to upper and lower extremities as needed <u>Contact layer</u> Mepitel® (Up to 10 days) or Mepilex® Border or Mepilex® Border Lite or Mepilex® or Mepilex® Lite	Mepitel® (Up to 10 days) or Mepilex® or Mepilex® Lite or Mepilex® Border or Mepilex® Border Lite or Alldress® or Comfeel® Plus	Mepilex® Border Lite or Mepilex® Lite or Apply 3M® No-Sting® Barrier (Daily) <u>Post Surgical</u> Mepilex® Border Post-Op <u>Radiation Dermatitis</u> Mepilex® Lite or Mepilex® Transfer
		Consider using Mepilex® Ag, Melgisorb® Ag or Aquacel® Ag when antimicrobial effect is desired						
Notations	- Use secondary dressing over Mepitel® - Use Mepitac® Tape on fragile skin. Secure dressings with Tubifast® of roll gauze. - For complex wounds consult the Wound Care Team - Date and time ALL dressings				- Utilize Mepilex® Heel or Mepilex® Border as needed for heel wounds and/or protection from shearing - Wear time for each dressing is up to 7 days unless otherwise noted - Cleanse wounds with normal saline or wound cleanser with each dressing change - Dressings with Safetac® technology do NOT require use of skin barrier products.			

Individuals with wound infection or those at high risk for infection may require more frequent changes as well as adjunctive antibiotic therapy. Before any healing process can begin, two critical steps must be taken as part of a well-defined management protocol:

1) The wound assessment and 2) Management of causative and contributing factors including unrelieved pressure, shear and friction, excessive moisture and altered nutritional status.

* Debridement of eschar may be contraindicated in some situations such as dry, fused, stable eschar. Debridement is indicated if signs/symptoms of infection are present.

† Creams or ointments may be applied over Mepitel® as indicated. Mepitel® may be left in place during wound cleansing and irrigation. Change secondary dressings as needed.

The suggested topical management options and change rates are the treatment choice of Walter Reed Bethesda and may not reflect the opinions of Mölnlycke Health Care or in the case of products manufactured by a company other than Mölnlycke Health Care, the manufacturer's recommended usage guidelines.

Fig. 6 Walter Reed wound care selection guide. (Courtesy of Walter Reed National Military Medical Center, Bethesda, MD.)

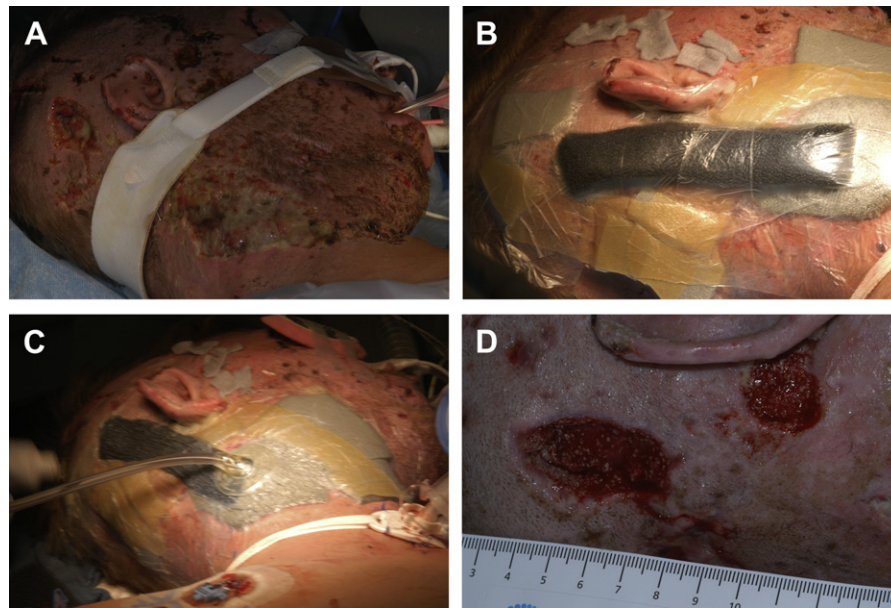


Fig. 7 Application for wound VAC. (A) Wartime shrapnel wound showing large avulsive areas of right postauricular head and neck. (B) Application of wound VAC dressing. (C) Wound VAC activation. (D) Forty-eight hours after first wound VAC application. Wound bed appears healthy with small focal areas of granulation forming.

- Dietitians
- Physiotherapists
- Occupational therapists
- Pharmacists
- Family and/or other caregivers

By targeting the wound from different aspects provided by the wound-care team, acute and chronic wounds can be better managed, leading to a quicker resolution of the wound.

Summary

There are many complex factors influencing wound healing. In addition, there are no absolutes on the type of dressing that should be used. The most effective dressing is one that promotes healing by decreasing the bioburden, provides a protected and moist environment, does not require frequent changing, and provides patient comfort. A provider must always assess the amount of wound drainage and depth of the wound to determine the appropriate dressing.

Summary

The process of wound healing is complicated and requires optimization of wound bed conditions locally through wound management and systemically through proper nutritional care. Although there are a variety of local and systemic factors that can adversely influence healing, the wound environment can be treated through proper dressings to decrease necrotic debris, bacterial load, and foreign bodies. In addition, maintaining or improving patient nutritional status will help the body to supply the necessary building blocks and cellular response for healing to take place.

Further readings

Argenta LC, Morykwas MJ. Vacuum-assisted closure: a new method for wound control and treatment. *Ann Plast Surg* 1997;38:563.

- Arnold M, Barbul A. Nutrition and wound healing. *Plast Reconstr Surg* 2006;117(Suppl 7):42S–58S.
- Attinger CE, Janis JE, Steinberg J, et al. Clinical approach to wounds: debridement and wound bed preparation including the use of dressings and wound-healing adjuvants. *Plast Reconstr Surg* 2006;117(Suppl 7):72S–109S.
- Broughton 2nd G, Janis JE. Wound healing: an overview. *Plast Reconstr Surg* 2006;117(Suppl 7):1e–S–32e–S.
- Brown KL, Phillips TJ. Nutrition and wound healing. *Clin Dermatol* 2010;28:432–9.
- Davis LF. Fluids, electrolytes and nutrition in the oral and maxillofacial surgery patient. *Oral Maxillofac Surg Clin North Am* 1996;4:3.
- Eagelstein W. Moist wound healing with occlusive dressings: a clinical focus. *Dermatol Surg* 2001;27(2):175–81.
- Ehrlich HP. The physiology of wound healing. A summary normal and abnormal wound healing processes. *Adv Wound Care* 1998;11(7):326–8.
- Fang JC, Chirag DN, Dym H. Nutritional aspects of care. *Oral Maxillofac Surg Clin North Am* 2006;18:1.
- Goldberg SR, Diegelmann RF. Wound healing primer. *Surg Clin North Am* 2010;90(6):1133–46.
- Kang D, Ellis E. Application of Vacuum-Assisted Closure Device in Maxillofacial Reconstruction. *J Oral Maxillo Surg* 2010;68(12):3037–42.
- Kavalukas S, Barbul A. Nutrition and wound healing: an update. *Plast Reconstr Surg* 2011;127(Suppl 1):38S–43S.
- Kerstein MD. The scientific basis of healing. *Adv Wound Care* 1997;10(3):30–6.
- Mc Callon ST, Knight CA, Valiulus P, et al. Vacuum-assisted closure versus saline-moistened gauze in the healing of postoperative diabetic foot wounds. *Ostomy Wound Manage* 2000;46(8):28–34.
- Morykwas MJ, Argenta LC, Shelton-Brown EI, et al. Vacuum assisted closure: a new method for wound control and treatment: animal studies and basic foundation. *Ann Plast Surg* 1997;38:553.
- Okan D, Woo K, Ayello EA, et al. The role of moisture balance in wound healing. *Adv Skin Wound Care* 2007;20(1):39–53.
- Posthauer ME, Dörner B, Collins N. Nutrition: a critical component of wound healing. *Adv Skin Wound Care* 2010;23:560–72.
- Rashad UM, Al-Gezawy SM, El-Gezawy E, et al. Honey as topical prophylaxis against radiochemotherapy-induced mucositis in head and neck cancer. *J Laryngol Otol* 2009;123:223–8.
- Sherman AR, Barkley M. Nutrition and wound healing. *J Wound Care* 2011;20:8.

- Song JJ, Salcido R. Use of honey in wound care: an update. *Adv Skin Wound Care* 2011;24(1):40–4.
- Stechmiller JK. Understanding the role of nutrition and wound healing. *Nutr Clin Pract* 2010;25:61.
- Velnar T, Bailey T, Smrkolj V. The wound healing process: an overview of the cellular and molecular mechanisms. *J Int Med Res* 2009;37(5): 1528–42.
- Wild T, Rahbarnia A, Kellner M, et al. Basics in nutrition and wound healing. *Nutrition* 2010;26:862–6.
- Williams JZ, Barbul A. Nutrition and wound healing. *Crit Care Nurs Clin North Am* 2012;24:179–200.
- Winter G. Formation of the scab and the rate of epithelisation of superficial wounds in the skin of the young domestic pig. *Nature* 1962;193:293.

Soft Tissue Trauma

Chris Crecelius, DDS

KEYWORDS

• Soft tissue trauma • Wound healing • Wound closure • Wound care

KEY POINTS

- Copious irrigation with normal saline is the only debridement and preparation needed for most soft tissue wounds before closure.
- Well-irrigated and debrided facial soft tissue wounds do not require antibiotics.
- Careful resuspension of soft tissue with approximation and eversion of the wound at the dermal level will provide superior esthetic outcomes.
- Explore soft tissue trauma carefully, keeping in mind the pertinent anatomy, so that vital structures are treated along with the closure of the wound.
- Follow the healing wound, and intervene in the first few weeks to modulate the healing process for the best outcome.

Soft tissue trauma is a commonly encountered sequel of head and neck trauma (Figs. 1–3). The injury may be limited to superficial structures or may be the harbinger of injury to deeper anatomic structures. The ability to accurately diagnose soft tissue injuries, manage their repair, and modulate the healing process will provide your patients a superior outcome.

Skin

Soft tissue injuries, by their nature, involve the overlying skin and/or mucosa of the head and neck. The skin is the largest organ of the body and is also the most abused. The skin is, however, remarkably resilient and has a high capacity to heal. It provides protection from the environment, regulates body temperature, prevents fluid loss, and prevents entry of pathogens. It is made up of 3 general layers: the epidermis, dermis, and subcutaneous connective tissue (Fig. 4). The epidermis is stratified squamous epithelium. It contains no blood vessels, minimal extracellular matrix, and few nerves. It does contain Langerhans cells, which are antigen-presenting cells of the immune system. It ranges in thickness from 0.05 mm over the eyelids to 1.5 mm over the soles of the feet. The epidermis is a waterproof, semipermeable membrane that turns over in about 40 days. Epidermal cells migrate through the 5 layers of the epidermis. They start at the basement membrane, which separates the epidermis from the dermis, and ultimately are desquamated at the skin's surface. The 5 cell layers are the basal cells (the only dividing cell layer), stratum spinosum, stratum granulosum, stratum lucidum, and stratum corneum.

Disclaimer: The views expressed are those of the author and not necessarily those of the Department of Defense, United States Navy, United States Navy Bureau of Medicine and Surgery, or the United States Navy Dental Corps.

Division of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, MD 20889, USA

E-mail address: Chris.E.Crecelius.mil@health.mil

Dermis

The dermis makes up about 90% of the thickness of skin. It supports the epidermis with its collagen matrix and contains the dermal appendages, such as hair follicles, sweat glands, and sensory organs. The dermis provides nutritional support to the epidermis with a robust vascular plexus. Local and regional flaps owe their viability to this vascular plexus. It is thinnest over the eyelids (0.3 mm) and thickest over the back (3 mm). Closure of soft tissue wounds occurs primarily at the level of the dermis because it has the necessary tensile strength to support suture/staple closure. Accurate reapproximation of the dermis with eversion of the wound edges provides the greatest chance for a cosmetic closure. The dermis is 70% collagen by dry weight and 2% elastin, with glycosaminoglycans taking up the intervening space. A 1-mm collagen strand has a static load capacity of 20 kg. The subcutaneous tissue is a variable layer of adipose and connective tissue that intervenes between the skin and underlying musculature/fascia or skeleton. This subcutaneous fat allows the skin to move somewhat independently of the underlying tissue.

Physical properties

Skin has biphasic deformation properties. There is a rapid initial extension and a slow secondary extension termed *creep*. The initial extensibility is caused by elastin being stretched and collagen aligning. Creep is stress relaxation and is caused by the gradual change in collagen bonding and displacement of water. If skin is put under tension, the force required to maintain a constant length decreases over time. This property of skin can be used to close avulsion defects and is particularly helpful for scalp wounds. Skin also has the property of resting skin tension lines (Fig. 5). Skin's strength and distensibility are directional, which is caused by collagen orientation. Lacerations from blunt trauma tend to follow the resting skin tension lines. These lines are in the direction that the skin is weakest,



Fig. 1 A 20-year-old woman status post s/p ejection from motor vehicle through windshield. Patient sustained lacerations, abrasions, and an avulsion injury to the right cheek.

that is, perpendicular to muscle pull and parallel to dermal collagen bundles. Wounds following these lines tend to heal with the best cosmetic outcome.

Healing

Skin and soft tissue heal in a regular pattern: an inflammatory phase, proliferative phase, and remodeling phase. The inflammatory phase is characterized by fibrin deposition and the start of epithelial cell migration under fibrin and over collagen. This phase lasts from 48 to 96 hours. The proliferative phase is characterized by fibroblast migration with production of extracellular matrix. It starts around the third to fourth day. Myofibroblasts contract the wound and macrophages clear



Fig. 2 Same patient seen in Fig. 1 after irrigation, debridement, and closure of lacerations in emergency department.



Fig. 3 Same patient seen in Figs. 1 and 2 after full-thickness skin graft from supraclavicular region and fat transfer to right cheek. Patient would likely benefit from dermabrasion to smooth skin contours and even skin tone.

debris. The remodeling phase begins with the decrement in fibroblast and macrophage number at around 3 weeks. At 6 weeks, the collagen synthesis and degradation is equalized. The final strength of the repaired tissue reaches 70% to 80% of the preinjury level (Boxes 1 and 2).

Wound types

Facial wounds can be one of several types but are frequently a combination. A simple laceration is similar to a surgical incision. These lacerations should be irrigated and closed with minimal undermining. Complex lacerations require a more detailed study of their pattern to return tissue to its original position and should be closed in a layered fashion, suspending tissue as required to remove tension from wound edges.

Abrasions are the removal of the epidermis and outer portion of the dermis. Carefully debride and clean abrasions and cover with antibiotic ointment. These wounds should not be allowed to dry out during their re-epithelialization. Re-epithelialization will occur from keratinocytes migrating from the wound edges and from adnexal structures.

Avulsive injuries involve the loss of soft tissue, including complete loss of the epidermis and dermis. These wounds may be treated with the use of wound vacuums to prepare the wound bed for grafting procedures and/or reduce the size of the wound. Wet to dry dressings may also be used to debride the wound and allow the formation of granulation tissue. Avulsive injuries may be closed by secondary intention, local flaps, regional flaps, or grafts (Figs. 6–8).

Puncture wounds have a small epidermal defect in comparison with their depth. The greatest challenge with puncture wounds is achieving adequate irrigation, which may require the use of catheters to reach the depth of the wound or may require opening the wound.

Contusions and blast injuries may produce extensive soft tissue damage, which is not readily apparent on initial presentation. These injuries compromise the microvasculature and, thus, the healing capacity of the soft tissue. Minimal manipulation is warranted with monitoring of the tissue to determine vitality and signs of infection (Figs. 9–11). Burns require specialized treatment and should be referred to a burn unit for treatment except for those limited in surface area and depth. Small, superficial burns can be treated like an abrasion with topical antibiotic ointments.

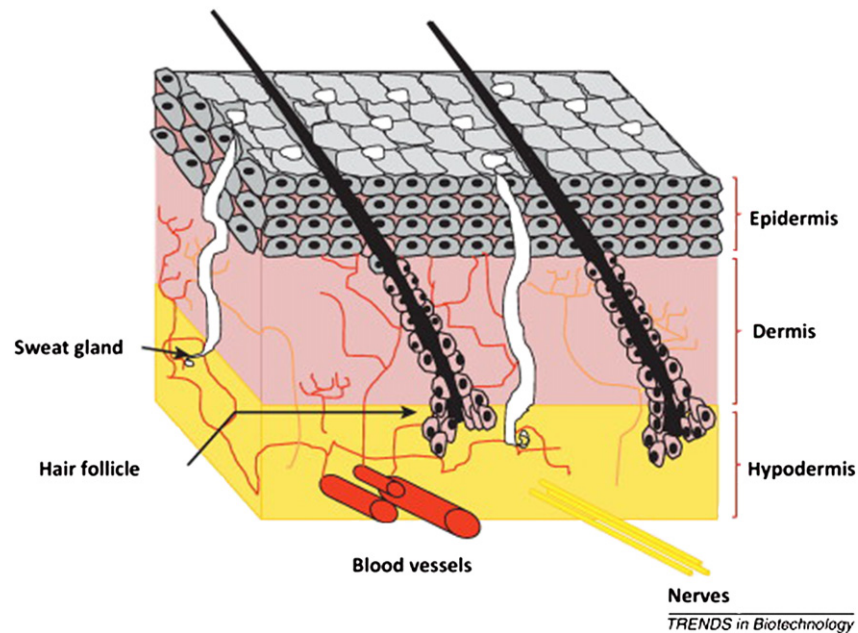


Fig. 4 The 3 layers of the skin and dermal appendages. (From Yildirim L, Thanh NT, Seifalian AM. Skin regeneration scaffolds: a multimodal bottom-up approach. *Trends Biotechnol* 2012;30(12):638–48; with permission.)

Approach

The approach to the treatment of soft tissue trauma is an iterative process. The examination of the wound begins the moment patients are first visualized. Further evaluation of the facial wounds is then deferred to manage patients' general medical condition following advanced trauma life-support protocol. Other than soft tissue trauma compromising the airway or resulting in life-threatening hemorrhage, treatment of facial trauma is delayed until patients are stabilized.



Fig. 5 The resting skin tension lines. (From McGillis ST, Lucas AR. Scar revision. *Dermatol Clin* 1998;16(1):165–80; with permission.)

Box 1. Factors that interfere with wound healing

- Local
 - Infection
 - Foreign bodies
 - Ischemia
 - Smoking
 - Radiation
 - Trauma
 - Cancer
 - Local toxins
 - Arterial insufficiency
 - Venous insufficiency
 - Hyperthermia
- Systemic
 - Inherited disorders
 - Nutritional deficiencies
 - Aging
 - Diabetes
 - Liver disease
 - Alcoholism
 - Uremia
 - Medications
 - Blood transfusions
 - Jaundice

Data from Lawrence WT. Clinical Management of Non-healing Wounds. In: Cohen IK, Diegelmann RF, Lindblad WJ, editors. Wound healing: biochemical and clinical aspects. Philadelphia: Saunders; 1992.

Box 2. Factors contributing to wound infections

Trauma
Malnutrition
Immune suppression
Arterial ischemia
Venous congestion
Lymphedema
Foreign bodies
Crush injury
Necrotic tissue
Denervation
Wound maceration
Obesity
Prolonged surgery
Age

Data from Achauer BM, Eriksson E. Plastic surgery: indications, operations, and outcomes. Part I principles and techniques. St Louis (MO): Mosby; 2000. p. 66.

History and physical

The history and physical was already begun during the Advanced Trauma Life Support Change (ATLS) surveys but continues after the patient's initial stabilization. Persistent facial hemorrhage should be controlled. Focused questions related to the facial soft tissue trauma should be pursued. Knowing the events surrounding the trauma will provide information on the mechanism, possible contamination, time since injury, and time since last oral intake. The patients' general medical condition will have an impact on the patients' wound-healing capacity.

Physical

The physical examination of the facial soft tissue trauma cannot be completed unless patients are lucid enough to respond purposefully to verbal commands and questions. It also cannot be completed until the wound is debrided and irrigated. The examination, debridement, irrigation, and hemostasis of the wound occur simultaneously. The examination must ascertain the level of function of the motor and sensory nerves and the extent of disruption of deeper structures, such as

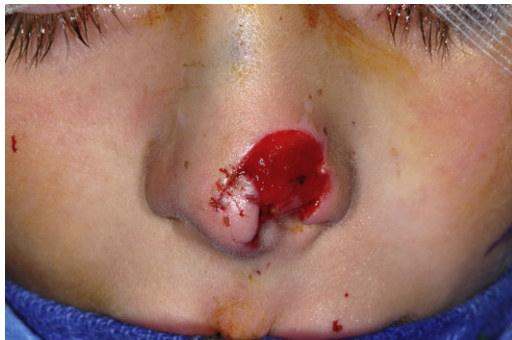


Fig. 6 Avulsion of left ala of nose, including skin and cartilage.

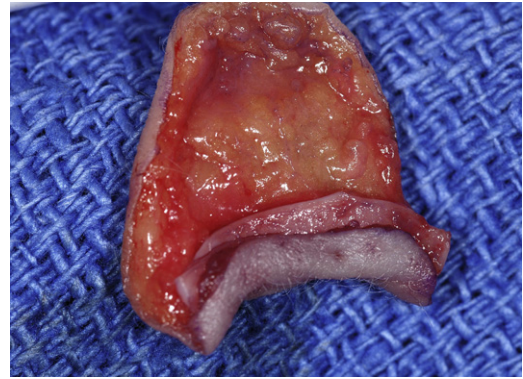


Fig. 7 Same patient as Fig. 6 showing composite graft from helix of ear.



Fig. 8 Same patient as Figs. 6 and 7 showing the inset composite graft from the ear to the left ala of the nose.



Fig. 9 M16 round to midface, a low-energy gunshot/blast type injury.

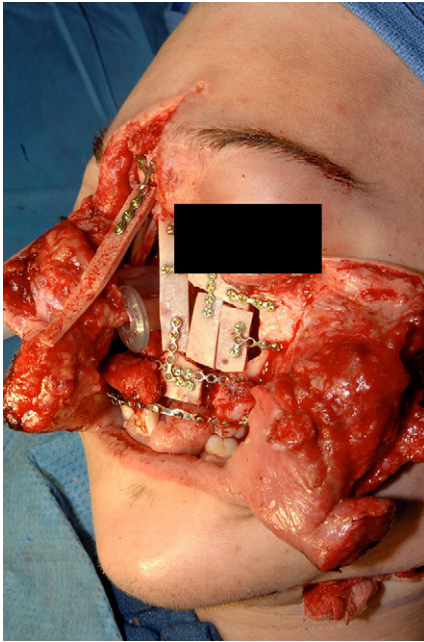


Fig. 10 Same patient seen in [Fig. 9](#) after primary calvarial bone grafting to replace missing midface skeleton.

salivary ducts, lacrimal apparatus, tarsal plates, external auditory canal (EAC), underlying bone, and so forth.

Irrigation and debridement

Maximum preservation of facial soft tissue should be attempted. Irrigation with copious normal saline and digital agitation will almost always adequately irrigate and debride the wound. A 1-L bottle with a single 18-gauge needle hole in the lid or with an irrigating tip attached provides the force necessary for



Fig. 11 Same patient seen in [Figs. 9 and 10](#) after primary closure and microvascular free flap to close avulsion injury to left cheek.

irrigation and debridement. A 60-mL syringe with an intravenous (IV) catheter attached may also be used. Pulsatile jet lavage should be reserved only for those wounds with gross contamination that are not adequately debrided by hand. Pulsatile jet lavage is damaging to soft tissue.

Betadine, povidone-iodine, chlorhexidine, and hydrogen peroxide are all damaging to soft tissue. The prep should be confined to intact skin except in cases of gross contamination, whereby the benefits of prep outweigh the harm. If saline alone is inadequate, 50% hydrogen peroxide and 50% saline mixture can be particularly useful for removing debris and dried blood. Saline irrigation within the first 24 hours of injury is effective in all wounds except those contaminated with pus.

Hemostasis

Controlling hemorrhage occurs at multiple points during the examination and debridement of wounds. Controlling hemorrhage may be done initially for gross hemorrhage but is often required again during debridement and irrigation. Direct pressure is the foremost modality. Local anesthetics with epinephrine may be useful but carry the risk of rebound hemorrhage. Tying vessels should be performed as either direct ties or stick ties for discrete vessels. Electrocautery should be reserved for situations when other measures fail because it is a determinate of poor outcomes for wound healing ([Box 3](#)).

Wound closure

There are several methods for closure of facial wounds. Sutures are the most frequently used. Staples, tape, and adhesive also may be considered in isolation or in combination with other techniques. Staples have the advantage of speed, wound edge eversion, and minimal inflammatory response. They are rarely used on the face but have great utility in hair-bearing areas because of the greater ease of placement and removal compared with sutures. Tape and adhesive are often used as adjuvants to suture closure or used alone on sub-centimeter superficial wounds, which are well approximated before closure. Tape is particularly tenuous as a sole method because it has variable longevity depending on its adherence to the skin. Wet, oily, or sweaty skin reduce tapes adherence. Use of skin adhesive can greatly prolong the adherence of tape. Skin inflammation from skin adhesive is not uncommon and may result in prolonged localized erythema and edema at the

Box 3. Determinates of poor wound healing

The statistically significant determinates of poor skin wound esthetics:

- Use of electrocautery
- Trauma to surrounding skin
- Incomplete wound apposition
- Wide wounds
- Wound infection

Data from Singer AJ, Quinn JV, Thode HC Jr, et al. Determinants of poor outcome after laceration and surgical incision repair. *Plast Reconstr Surg* 2002;110(2):429–35.

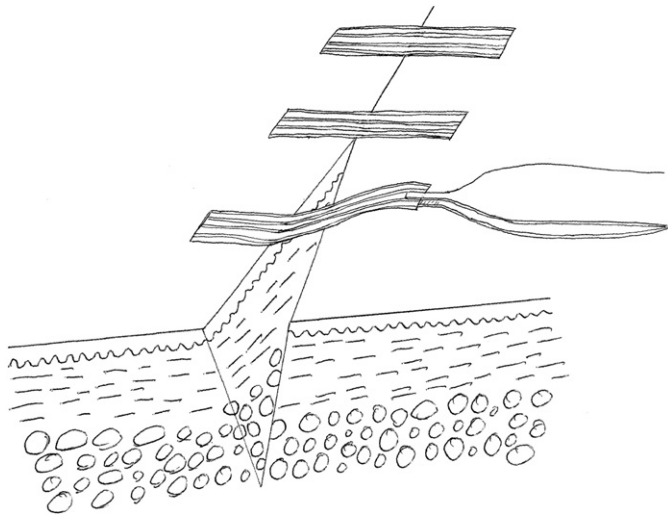


Fig. 12 Tape closure of a wound.

site of application. Tape can be very helpful in reducing tension across a wound after suture closure if placed with adhesive and while pulling the 2 wound edges together manually. Adhesive may be the quickest method for closing a wound, which can be useful in pediatric and uncooperative patients; but the adhesive usually gets into the wound, preventing direct soft tissue approximation (Fig. 12).

Suture

Suturing is the most common method for facial wound closure. Wound edges must be accurately approximated and everted for the most cosmetic outcome, which suturing facilitates. There are numerous options for suturing. In general, the best suture will have low tissue reactivity, will resorb when the tissue gains adequate strength to support itself, and will be a monofilament. Low reactivity and resorbability tend to be apposing requirements. Monofilaments tend not to handle as well as braided sutures. Thus, there have to be compromises made in choosing the best suture. Deep dermal sutures are frequently poliglecaprone (monocryl [Ethicon, Somerville NJ]: monofilament, knots less securely, resorbable, lower tissue reactivity) or polyglactin (Vicryl [Ethicon, Somerville NJ]: braided, knots more securely, resorbable, higher tissue reactivity). Skin

sutures are frequently polypropylene or nylon (monofilament, minimal acute inflammatory response, nonabsorbable) or gut (monofilament, moderate inflammatory response, absorbable). The use of gut may be considered when there is a low esthetic demand or when removal is problematic, such as in the pediatric population or poorly compliant adults. Polydioxanone (PDS [Ethicon, Somerville NJ]: slowly absorbing monofilament) is very useful for resuspending soft tissue to take the tension off wound edges during healing (Table 1).

Suture technique

There are a variety of techniques for suturing, some of which are illustrated here. The simple interrupted suture and running suture are the most commonly used, but the other techniques should be considered for their improved ability to take tension off the wound margins and evert the wound edges (Figs. 13–17).

Timing of wound closure

In general, closure of soft tissue wounds is best done as soon as possible after injury. If closure can be accomplished before significant edema begins, the closure is easier and more accurate. Hyaluronidase may be injected into edematous tissue and the edema may be displaced by application of direct pressure which may aid in wound approximation. Closure of soft tissue wounds within 12 hours of injury is best and closure within 6 hours is better. Successful closure of soft tissue wounds, without freshening wound edges, has been accomplished up to 48 hours after injury.

Wound care

Facial wounds should be covered in antibiotic ointment or other ointment and not allowed to dry out or scab. The ointment should be removed on a daily basis with soap and water. Systemic antibiotics are not routinely indicated for non-contaminated, adequately irrigated/debrided wounds. A tetanus vaccination booster should be considered if not done in the last 5 years. Crusting can be removed with hydrogen peroxide and water if soap and water alone are ineffective. Skin sutures should be removed in 5 to 7 days to reduce scarring from the suture. Consideration should be given to the use of tape to continue to remove tension from wound edges for an additional week. A good deep suture technique will reduce the

Table 1 Suture types

Name	Filament	Relative Tensile Strength (5 Highest)	Strength Profile	Absorption Profile	Tissue Reaction (5 Highest)
Fast, chromic gut	Virtual monofilament	2	75% 5–14 d	79–90 d via proteolysis	5
Silk	Braided	1	Progressive loss over 1 y	Sig at 2 y proteolysis	4
Poliglecaprone (Monocryl)	Monofilament	4	20%–30% 2 wk	90–120 d hydrolysis	2
Polydioxanone (PDSII)	Monofilament	4	70% 3 wk	180–210 d hydrolysis	2
Polyglactin (Vicryl)	Braided	4	64% 2 wk	56–70 d hydrolysis	3
Polyglycolic acid (Dexon [Covidien - Mansfield, MA])	Braided	4	35% 2 wk	90–120 d hydrolysis	3
Nylon/Ethilon	Monofilament	3	81% 1 y	Stable at 2 y	2
Polypropylene	Monofilament	2	—	None	1

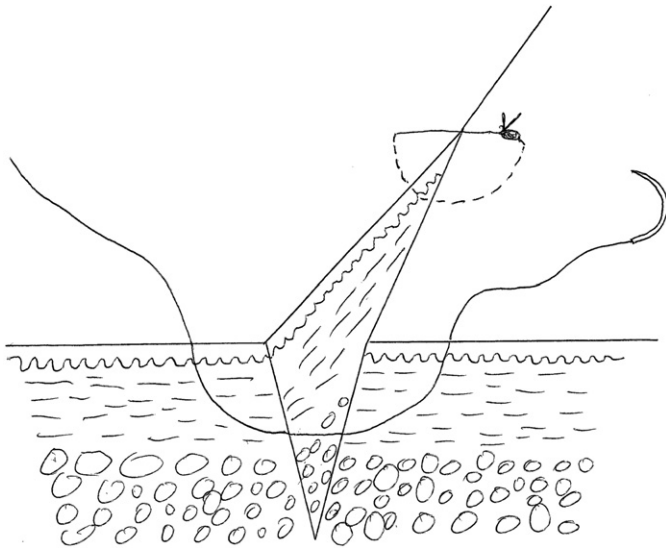


Fig. 13 Simple interrupted suture.

requirement for skin surface methods of reducing wound tension. Ointment without antibiotics should be used after the first week because of skin reactions to antibiotics. Triamcinolone and 5-fluorouracil injections may be considered for exuberant scars after the first 2 weeks. Silicone sheeting applied directly to the wound after epithelialization may also improve the outcome of wound closures. Laser therapy may aid in the healing process and reduce erythema. Dermabrasion, chemical peel, or laser resurfacing may also improve the contour and blend wound closures with the surrounding skin (Figs. 18–20). Excision and reclosure of scars may be considered to improve cosmesis. Tretinoin and hydroquinone creams may be helpful in cases of hyperpigmentation or hypopigmentation along with the use of topical hydrocortisone. Fat transfer, subcision, and/or fillers may also be helpful in improving contour.

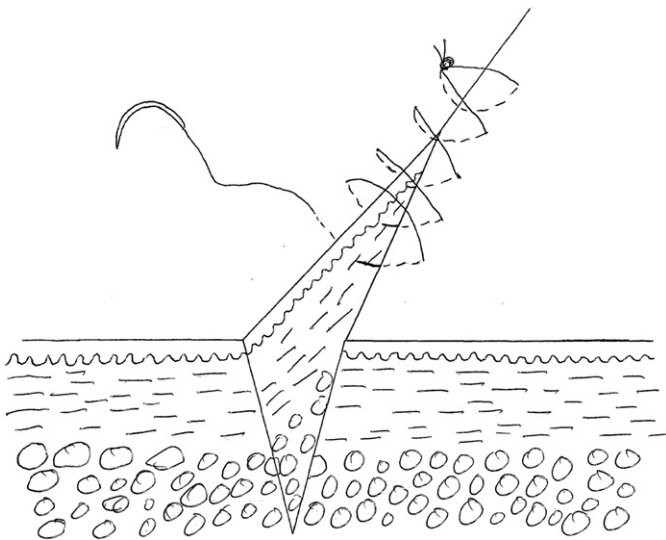


Fig. 14 The running suture is useful for longer lacerations and helps to even the tension across the wound over the length of the suture.

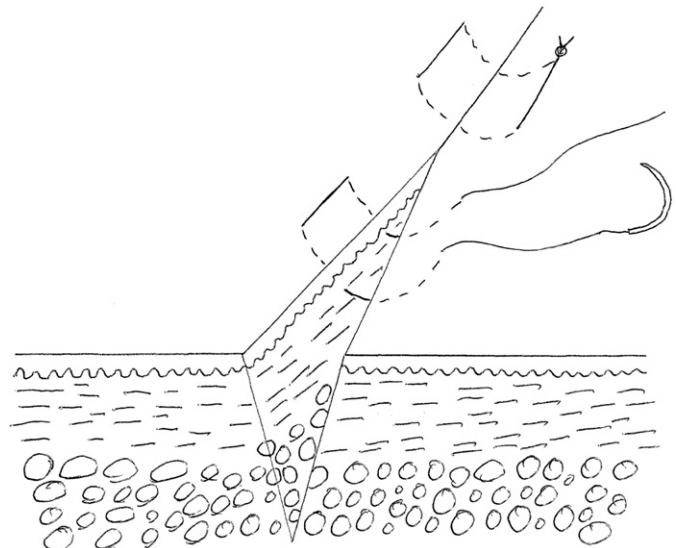


Fig. 15 The horizontal mattress suture is good for everting wound edges. Interrupted/running suture may be required to fine-tune the approximation of the wound edges within the confines of the horizontal mattress suture.

Special situations

Bites

Patients suffering animal and human bites may suffer from puncture wounds, which are more difficult to adequately irrigate. Antibiotics from the penicillin family are routinely prescribed. Augmentin is recommended, although penicillin has been shown to be equally efficacious. Clindamycin may be

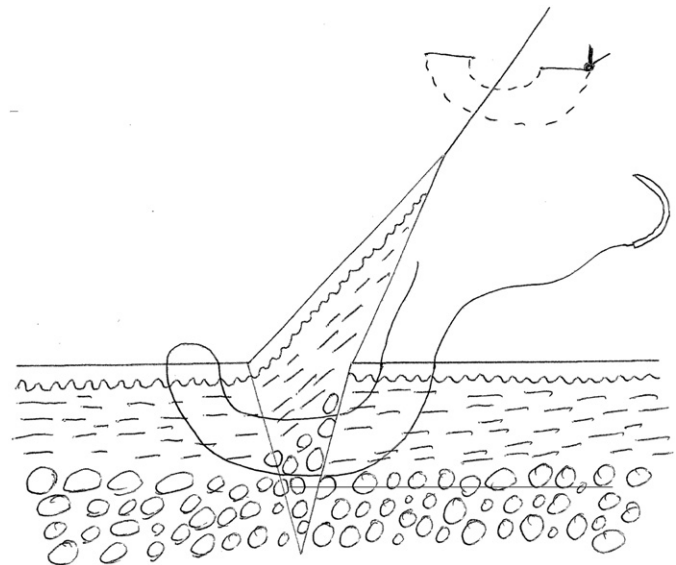


Fig. 16 The vertical mattress suture everts wound edges and also moves the tension further away from the wound margin. This technique is useful when significant force is required to close a wound such as may be found for a scalp wound. The technique spreads the suture tension over a larger area of skin away from the wound margin, which might result in tearing of the skin otherwise. Padding may be placed under the suture loops to further dissipate tension.

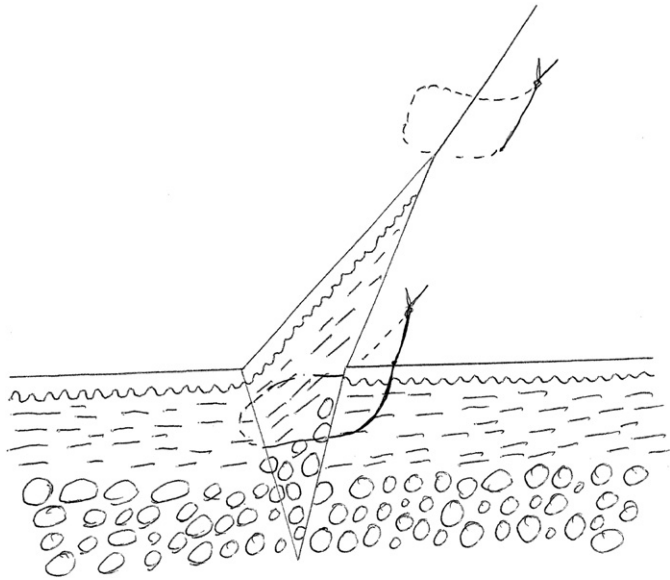


Fig. 17 Half buried horizontal mattress. This technique is useful for the triangular corners of wounds. It avoids piercing the skin and superficial dermis in an area of compromised blood supply.

used if patients are allergic to penicillin. There is not good evidence, however, that antibiotics affect the outcome of open wounds. The organisms of concern are *Pasteurella multocida*, *Staphylococcus aureus*, *Streptococcus viridians*, and *Capnocytophaga canimorsus*. For human bites, the infecting organisms are often *Eikenella corrodens*. Tetanus and rabies vaccines should be considered (Figs. 21 and 22).

Gunshot wounds/blast injuries

Gunshot wounds are characterized by a crush injury at the site the projectile enters the tissue, with a permanent cavity

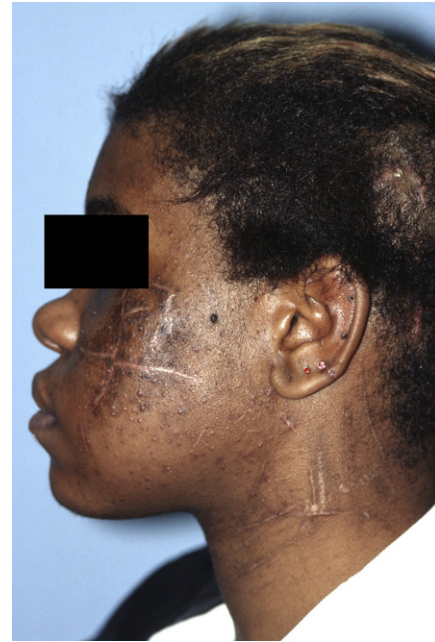


Fig. 19 Same patient as Fig. 18 five weeks after primary closure of left cheek wound, including approximation of parotid duct.

developed by direct tissue destruction from the path of the projectile. There is also a temporary cavity formed by the sonic/supersonic pressure shock wave of the projectile as it passes through the tissue. This shock wave damages the microvasculature. Blast injuries may have multiple projectiles associated with the shock wave. There may also be burns associated with blast and gunshot wounds. For low-energy wounds, the wounds should be copiously irrigated; but minimal to no tissue debridement may be necessary. The wounds are



Fig. 18 Patient status post motor vehicle crash with left cheek lacerations and Fitzpatrick V/VI skin.



Fig. 20 Same patient as Figs. 18 and 19 five weeks after dermabrasion of face, showing improved consistency of skin tone. Patient would benefit from continued hydrocortisone/tretinoin/hydroquinone topical treatment of facial skin.

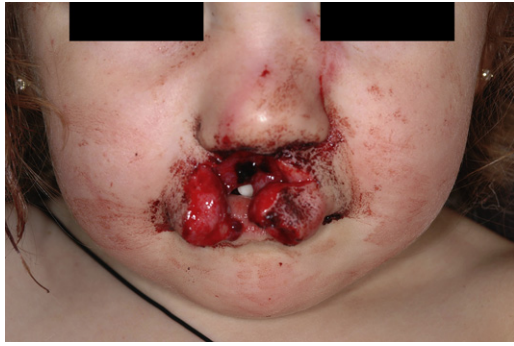


Fig. 21 Pediatric patient who suffered a bite to her upper lip from a dog.

then closed and followed for development of areas of necrosis. High-energy wounds have an evolving pattern of tissue loss caused by soft tissue and hard tissue devascularization. These wounds must be carefully debrided of foreign material with maximum preservation of soft tissue. Foreign material may act as a nidus for infection or produce tattooing. Pulsatile jet lavage may be beneficial during initial debridement for grossly contaminated wounds. Wet to dry dressings or wound vacuum treatment is then initiated. The wound is debrided every 24 to 36 hours until stable, which may take 3 to 10 days. Debridement is performed only of tissue that is obviously necrotic. The goal is to minimize soft tissue manipulation and maintain as much soft tissue as possible. After a stable condition has been achieved, the bone is reduced and rigidly fixated. The soft tissue is then closed over the skeleton. Consideration is given to replacing missing hard and soft tissue as needed. Monitor the healing process and intervene to modulate as necessary. Consider secondary procedures to improve function and esthetics. Broad-coverage antibiotics should be initiated as soon after the trauma as is possible (within the first hour). Delay in starting antibiotic prophylaxis increases the incident of wound infections (Figs. 23 and 24).

Facial nerve/peripheral nerve

Injuries to the cheeks and temple may involve the facial nerve. Branches of the nerve posterolateral to the lateral canthus of the eye are amenable to reapproximation. There must be a high index of suspicion based on the area and depth of injury, corroborated by the cranial nerve examination on cooperative patients, and careful exploration of the wound to find and



Fig. 22 Same patient as Fig. 21 after irrigation and primary closure of upper lip bite wound.



Fig. 23 Patient status post improvised explosive device blast with extensive facial soft tissue blast affected skin, which was treated with debridement of foreign bodies, wet to dry dressings, and antibiotic ointment.



Fig. 24 Same patient as Fig. 23 treated with wound vacuum after initial days of wet to dry dressings and antibiotic ointment. The patient later had split-thickness skin grafts to areas of avulsed skin, but the area that required grafting was much reduced after the use of the wound vac.



Fig. 25 Right cheek laceration involving the facial nerve and parotid duct. Wound has been irrigated and prepared for approximation of facial nerve branches. The parotid duct has been cannulated.

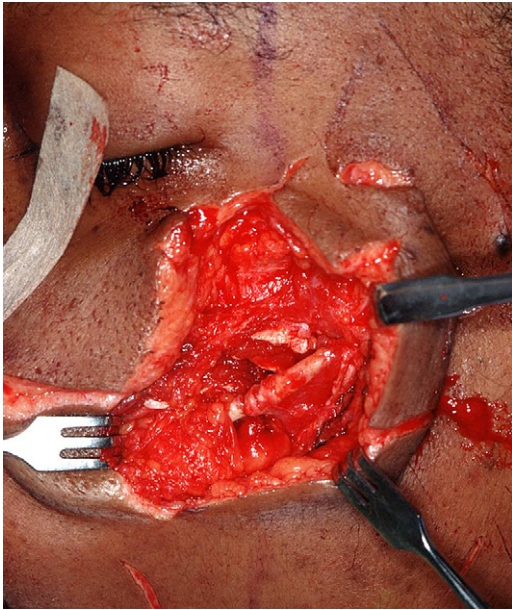


Fig. 26 Transected parotid duct identified in a left cheek laceration and approximated.

reapproximate the facial nerve. The best time to repair the nerve is immediately and with the use of magnification (Fig. 25). Repair should be attempted within the first 3 weeks of injury if discovered late. The lingual nerve and branches of the trigeminal nerve, among others, may also be injured. Injury to the lingual and trigeminal nerves will not be associated with any clinical signs, and discovery relies on the patient's symptoms. Repair of these nerves may be delayed 2 to 3 weeks to allow for surgical planning and consent if access will require additional incisions or osteotomies.

Salivary ducts/glands

To prevent a sialocele, disruption in the salivary gland capsule or duct must be recognized and closed in a watertight manner. The

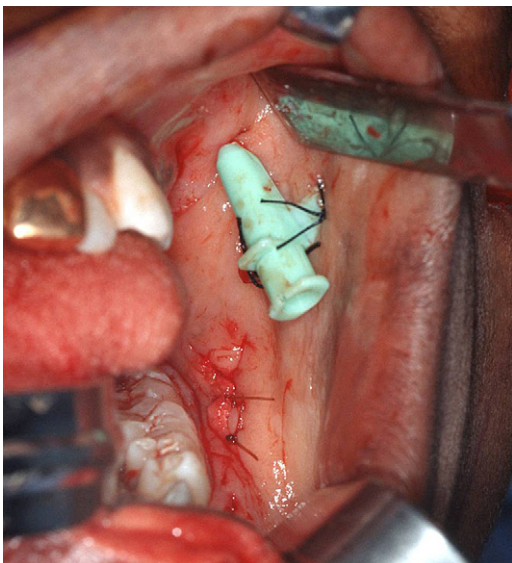


Fig. 27 Catheter sutured in place in the left cheek to stent open the parotid duct after transection repair.



Fig. 28 Nasal laceration with laceration of left lower lateral cartilage.

parotid capsule must be identified and the area explored for facial nerve transection. If transection of the parotid duct is ascertained from the clinical examination, the duct may be reapproximated under magnification and stented open for 2 weeks to allow for re-epithelialization of the duct (Figs. 26 and 27). Alternatively, the proximal stump of the parotid may be rerouted as in a sialodochoplasty or ligated and the gland allowed to atrophy. Antisialogogues or Botox may be used to reduce salivary flow. The sublingual and submandibular glands may be removed as treatment for damage to their capsule or duct.



Fig. 29 Same patient as Fig. 28 after approximation of lower lateral cartilage and closure of skin and mucosa.



Fig. 30 Patient after significant facial trauma who developed nasal stenosis from scarring associated with the healing process.

Nose

Concerns with soft tissue trauma to the nose include the development of a septal hematoma, transaction/dislodgement of cartilages, and nasal stenosis. A septal hematoma may result in destruction of septal cartilage and necrosis of the overlying mucosa with development of a septal perforation. The septum should be examined and the hematoma drained when identified. Transected cartilages should be reapproximated with as few monofilament sutures as possible and the skin and mucosa closed over the cartilage. Cartilages should be repositioned into their normal anatomic position (Figs. 28 and 29). Areas of mucosal damage may lead to internal nasal scarring with narrowing of the nasal airway caused by synechia or nasal stenosis. Internal nasal packing can help prevent synechia. To prevent nasal stenosis, rigid stenting of the nares may be required for 6 weeks. Modified nasal trumpets or custom nasal stents should be fabricated, which allows patients to remove the stent, clean it, and replace it on a daily basis during the healing process (Figs. 30 and 31).

Ear

Laceration of the auricle frequently involves the cartilage of the ear. The cartilage should be sutured with as few clear monofilament sutures as is required to reapproximate the wound. The external ear should be monitored for hematoma formation and bolster pressure dressing used if indicated. The external auditory canal must be examined and lacerations stented to prevent stenosis. The tympanic membrane and

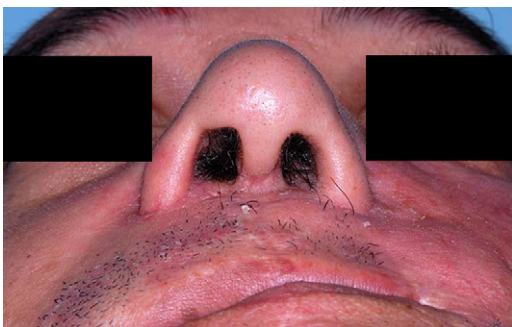


Fig. 31 Same patient as Fig. 30 who underwent reconstruction of the anterior nasal spine/nasal base, removal of internal nasal scarring, and cartilage grafting to support the nares. The patient was not completely compliant with the use of the nasal stent and has redeveloped a degree of nasal stenosis.



Fig. 32 Ear laceration. Cartilage should be approximated with as few clear monofilament sutures as required to hold it in position. The skin should then be closed and bolsters applied to prevent hematoma formation.

middle ear should also be examined for disruption and treated by the appropriate specialist (Fig. 32).

Eyelids

Lacerations of the eyelids and periorbital soft tissues should prompt a careful examination of the globe. Through-and-through lacerations may be approximated from inside to out with as few buried knot resorbable sutures as possible on the



Fig. 33 Eyelid laceration. The lacrimal ducts should be cannulated and assessed for patency.



Fig. 34 Through-and-through lip laceration that crosses the vermilion boarder. Accurate alignment of the vermilion boarder is important for a good esthetic outcome.

conjunctival surface and careful alignment of the gray line and tarsal plate. Canthal attachments should be examined and fixated as needed (Fig. 33).

Lacrimal apparatus

Injury to the medial third of the eyelids may result in injury to the lacrimal apparatus. The lacrimal puncta and ducts must be identified, cannulated, and approximated over polyethylene tubing, which is left in place for 2 weeks.

Lips

One of the big concerns with lips is alignment of the white role/vermillion boarder. Misalignment of the edge of the lip is noticeable to the casual observer. Lips are closed from the mucosal surface to the skin, with a watertight closure on the mucosal aspect. Avulsive injuries may be closed primarily if less than one-quarter of the upper lip or less than one-third of the lower lip length are missing. Defects exceeding this dimension are usually best treated with local flaps, such as an Abbe flap (Figs. 34–36).

Neck

The neck contains critical structures, such as the trachea, esophagus, great vessels, and nerve trunks. Clinical examination should be augmented with computed tomography (CT) or magnetic resonance (MR) imaging, and consideration should be



Fig. 35 Development of an Abbe flap to correct a defect in the upper lip.



Fig. 36 Same patient as Fig. 35 after inset of the Abbe flap. The healed flap before transaction of the vascular pedicle can be seen in Fig. 30. The final outcome of this flap can be seen in Fig. 31.

made for CT angiography or MR angiography. Endoscopic evaluation of the larynx, trachea, and esophagus should be considered. Neck wounds should be selectively explored. Consideration for stabilization of the cervical spine should be made early in the evaluation and treatment of the trauma patient.

Further readings

- Achauer BM, Eriksson E, editors. Plastic surgery: indications, operations and outcomes. St Louis (MO): Mosby; 2000.
- Aston SJ, Beasley RW, Thorne CH, editors. Grabb and Smith's plastic surgery. 5th edition. Philadelphia: Lippincott-Raven; 1997.
- Berk WA, Osbourne DD, Taylor DD. Evaluation of the 'golden period' for wound repair: 204 cases from a Third World emergency department. *Ann Emerg Med* 1998;17:496–503.
- Broughton G 2nd, Janis JE, Attinger CE. Wound healing: an overview. *Plast Reconstr Surg* 2006;117:1e-S.
- Byrnside V, Glasgow M, Gurunluoglu R. The vacuum-assisted closure in treating craniofacial wounds. *J Oral Maxillofac Surg* 2010;68:935–42.
- Ethicon wound closure manual. Somerville (NJ): Ethicon Inc.; 2004.
- Fonseca RJ. Oral and maxillofacial surgery. 2nd edition, vol. 2. Saunders Elsevier; 2009.
- Fonseca RJ. Oral and maxillofacial trauma. 3rd edition. vol. 2. Elsevier.
- Hollander JE, Richman PB, Werblud M, et al. Irrigation in facial and scalp lacerations: does it alter outcome? *Ann Emerg Med* 1998;31:73–7.
- Jones JS, Gartner M, Drew G, et al. The shorthand vertical mattress stitch: evaluation of a new suture technique. *Am J Emerg Med* 1993;11:483–5.
- Lee RH, Gamble WB, Mayer MH, et al. Patterns of facial laceration from blunt trauma. *Plast Reconstr Surg* 1997;99:1544–54.
- Messi G, Marchi AG. Evaluation of skin laceration repair by tissue adhesive in the pediatric emergency room. *Panminerva Med* 1992;34:77–80.
- Miloro M. Peterson's principals of oral and maxillofacial surgery. 2nd edition. Hamilton (Canada): BC Decker; 2004.
- Peacock EE. Wound repair. 3rd edition. Philadelphia: WB Saunders; 1984.
- Powers DV. Maxillofacial trauma treatment protocol. *Oral Maxillofac Surg Clin North Am* 2005;17(3):341–55.
- Ramasamy A, Hill AM, Clasper JC. Improvised explosive devices: pathophysiology, injury profiles and current medical management. *J R Army Med Corps* 2009;155(4):265–72.
- Margues de Medeiros I, Sacanato H. *Clin Evid* 2003 Jun;(9):2162–6.
- Sanders B, Andrews J, Akers P, et al. Management of wound break down after primary repair of facial lacerations. *J Oral Surg* 1974;32:531–4.
- Singer AJ, Quinn JV, Thode HCJ, et al. Determinants of poor outcome after laceration and surgical incision repair. *Plast Reconstr Surg* 2002;110:429–35.

Characterization and Management of Mandibular Fractures

Lessons Learned from Iraq and Afghanistan

David I. Tucker, DDS ^{a,*}, Michael R. Zachar, DDS ^{b,c}, Rodney K. Chan, MD ^d, Robert G. Hale, DDS ^a

KEYWORDS

• Mandible • Fracture • Combat-related injury

KEY POINTS

- Proper treatment cannot be completed without an accurate diagnosis.
- Whenever possible, occlusion should be used to guide reduction.
- Anatomic reduction is the goal.
- In complex fractures, maintain large segments of bone and obtain soft tissue coverage.

Introduction

The ongoing wars in Iraq and Afghanistan have provided the oral and maxillofacial surgeon unique challenges in reconstructing and restoring function to these soldiers with complex facial injuries. Indeed, injuries that were unsurvivable in previous conflicts are now commonplace because of early surgical intervention, body armor, and rapid evacuation. This article examines the history, etiology, diagnosis, classification, treatment, and complications of mandibular fractures, with emphasis on the challenges in treatment of facial injuries associated with blast and penetrating injuries common in Iraq and Afghanistan.

History

Archeological evidence shows humans have survived complex mandibular fractures long before they were documented in written history.¹ The first writings appeared as early as 1650 BC, but it was Hippocrates who first developed the concept of reapproximation and immobilization in 400 BC.² The development of

our current practice has been slow, with the importance of occlusion first introduced in 1180.³ Certainly, until the late 19th century, fixation of fractures centered on monomaxillary wiring and external bandages.

Hippocrates said, "War is the only proper school for a surgeon." Indeed, many major advances in treating maxillofacial injuries have arisen from conflicts.

The United States Civil War resulted in the next major technological advance in treating mandibular fractures—the use of interdental splints and intermaxillary fixation.⁴ Thomas Brian Gunning⁴ showed the importance of dentistry in treating these fractures by restoring occlusion with vulcanite splints.

During World War I, further advancement in treatment was pioneered by Kazanjian, who began wiring segments of bone together in combination with intermaxillary fixation.⁵ The external fixator, developed in 1936, was widely in use during World War II and continues to be useful in complex mandibular fractures.⁵ Internal fixation as we know it would be impossible without the development of safe antibiotics in the 1940s.

From the 1960s to the present, the focus in treatment of mandibular fractures has focused on internal fixation. Early treatment focused on large bulky plates placed through extraoral incisions. Over time, technology has resulted in smaller plates placed through intraoral incisions, which are effective in many fractures.^{6–8} Current technology seems focused on resorbable plates composed of copolymers of D- and L-lactic acid. Titanium and biodegradable miniplates are now often used in place of larger reconstruction bars with good success.^{8,9}

Combat-related maxillofacial injuries are primarily caused by explosives. The mandible is most commonly injured, with open fractures 3 times more common than closed fractures. These injuries are difficult to classify, and treating these often avulsive, penetrating, and burn injuries presents new challenges in our field (Fig. 1).^{10,11}

The wars of Iraq and Afghanistan will continue to challenge our capabilities as oral and maxillofacial surgeons. These

Disclaimer: The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army, Air Force or Defense.

^a Dental and Trauma Research Division, U.S. Army Institute of Surgical Research, 3698 Chambers Pass, Building 3611, Fort Sam Houston, TX 78234-6315, USA

^b San Antonio Military Medical Center, Fort Sam Houston, TX, USA

^c Department of Oral and Maxillofacial Surgery, Brooke Army Medical Center, Fort Sam Houston, TX 78234, USA

^d U.S. Army Institute of Surgical Research, 3698 Chambers Pass, Building 3611, Fort Sam Houston, TX 78234-6315, USA

* Corresponding author.

E-mail addresses: David.tucker@amedd.army.mil, tuckerdds@gmail.com

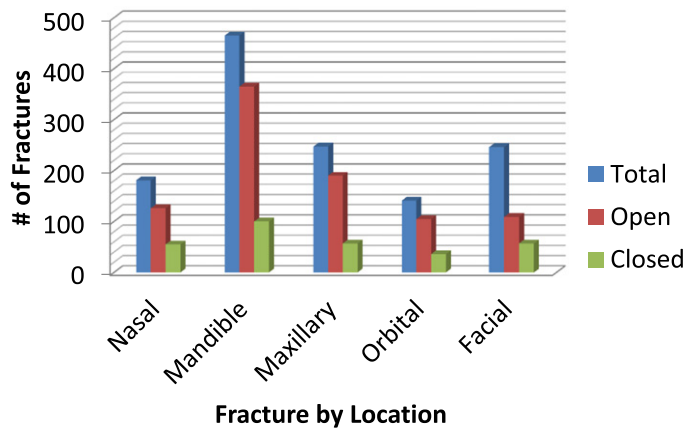


Fig. 1 Distribution of combat-related craniomaxillofacial fractures in Operation Iraqi Freedom and Operation Enduring Freedom from October 2001 to December 2007. (Adapted from Lew TA, Walker JA, Wenke JC, et al. Characterization of craniomaxillofacial battle injuries sustained by United States service members in the current conflicts of Iraq and Afghanistan. *J Oral Maxillofac Surg* 2010;68(1):3–7; with permission.)

injuries often involve complex burns and devastating tissue loss. The care of these patients will usually require multiple surgeries and coordination with critical care, neurosurgery, plastic surgery, anesthesia, and frequently psychiatry, speech therapy, and prosthodontics. Advances in regenerative medicine, wound healing, and even composite tissue allografting may be the future of treatment in these demoralizing injuries.

Etiology of mandibular fractures

Early analysis of data by Zachar and Lew (Zachar MR, Labella C, Kittle CP, et al. Characterization of mandible fractures incurred from battle injuries in Iraq and Afghanistan from 2001–2010. Submitted to *J Oral Maxillofac Surg*) shows that the current system of facial injury classification is inadequate. The current coding system is insufficient in reporting the amount of tissue loss, burns, and atypical fracture patterns found in war injuries. A better system of reporting these injuries may improve care and decrease the number of procedures for these patients.

Mandible fractures are among the most frequently encountered types of facial injury in developed and undeveloped countries. The cause is usually by violent crime (assault) or motor vehicle accidents. Classification varies but minimally should include number of fractures, relationship to external environment, presence of teeth, and location (Figs. 2–5).

When comparing battle injuries in Afghanistan and Iraq with civilian trauma, fractures involving the mandibular body and angle are significantly higher in the battle-injured population (Fig. 6). This is because of the nature of blast injury forces compared with those of blunt trauma (Zachar MR, Labella C, Kittle CP, et al. Characterization of mandible fractures incurred from battle injuries in Iraq and Afghanistan from 2001–2010. Submitted to *J Oral Maxillofac Surg*).

Fracture classification by anatomic region

- Midline—fracture between central incisors³
- Parasymphyseal—fractures occurring within the area of the symphysis

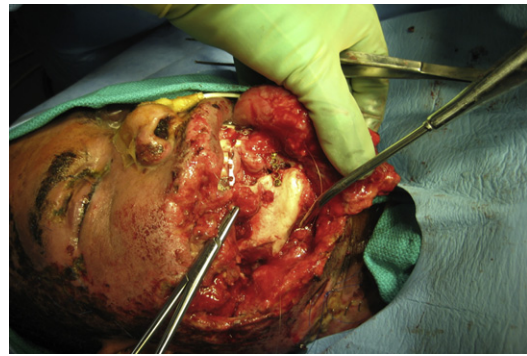


Fig. 2 Complex facial injury with avulsive tissue loss. Many combat injuries result in burns, significant tissue loss, and exposed bone.



Fig. 3 Avulsive injury caused by explosive. Note loss of commisure, upper and lower lip defects, and burn eschar.

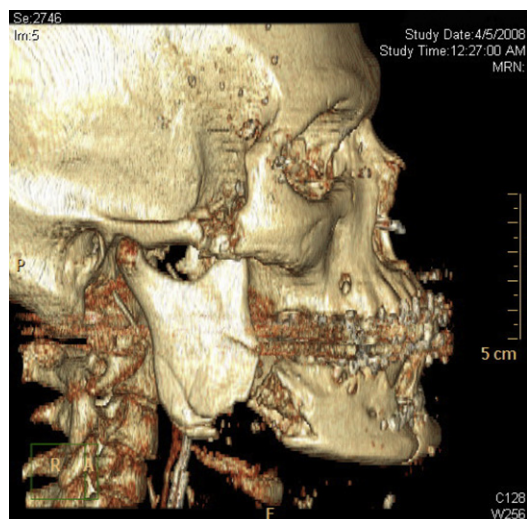


Fig. 4 Three-dimensional CT of patient in Fig. 2. Note avulsion of large segment of mandibular body.

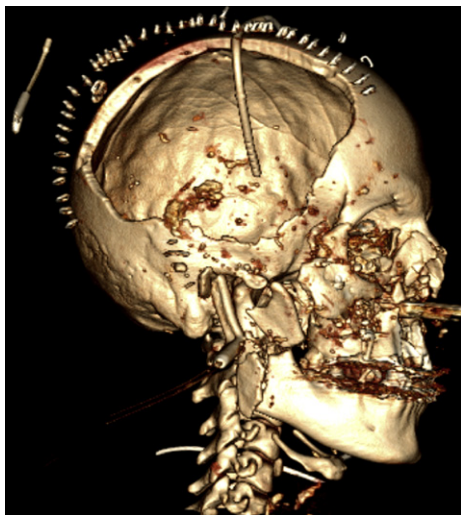


Fig. 5 Three-dimensional CT of patient in Fig. 3. Note extensive comminution typical with explosive injury to the face.

- Symphysis—bounded by vertical lines distal to the canine teeth
- Body—from the distal symphysis to a line coinciding with the alveolar border of the masseter muscle
- Angle—triangular region bounded by the anterior border of the masseter muscle to the posterosuperior attachment of the masseter muscle
- Ramus—bounded by the superior aspect of the angle to 2 lines forming an apex at the sigmoid notch
- Condylar process—area of the condylar process superior to the ramus region
- Coronoid process—includes the coronoid process of the mandible superior to the ramus region
- Alveolar process—the region that would normally contain teeth (Fig. 7)

Common descriptive terms of fractures

- Simple (Closed)—fracture without wound open to external environment¹²

- Compound (open)—fracture in which an external wound, involving skin, mucosa, or periodontal membrane, communicates with the break in the bone
- Comminuted—fracture in which the bone is splintered or crushed
- Greenstick—fracture in which only one cortex of the bone is fractured
- Pathologic—fracture occurring due to presence of disease
- Multiple—2 or more lines of fracture on the same bone not communicating with each other
- Impacted—a fracture in which one fragment is firmly driven into another
- Atrophic—fracture resulting from atrophied bone
- Indirect—a fracture at a point distant from the site of injury
- Complicated (complex)—fracture with considerable injury to the adjacent soft tissue or adjacent parts, may be simple or compound

Shetty and colleagues¹³ recognize the lack of objectivity and standardization with our current methods of characterizing mandibular fractures. They have developed the UCLA Mandible Injury Severity Score to numerically classify the severity of injury and guide treatment. Unfortunately, this analysis eliminated complex injuries like gunshot wounds, so its use for characterizing battle injuries would be limited.

Fractures involving the condyle should be considered separately. Multiple classification systems have been proposed, but generally they are classified as intracapsular, extracapsular, or subcondylar. Degree of displacement and comminution will generally dictate treatment.

Diagnosis/evaluation

A thorough history and physical examination are performed once the airway is secured and the patient is hemodynamically stable. The history can provide clues to the types of injuries expected, changes in occlusion, and medical issues that may influence treatment.

Palpation of the condyles and inferior border of the mandible will find obvious fractures, whereas the intraoral examination will find malocclusion, missing teeth, range of

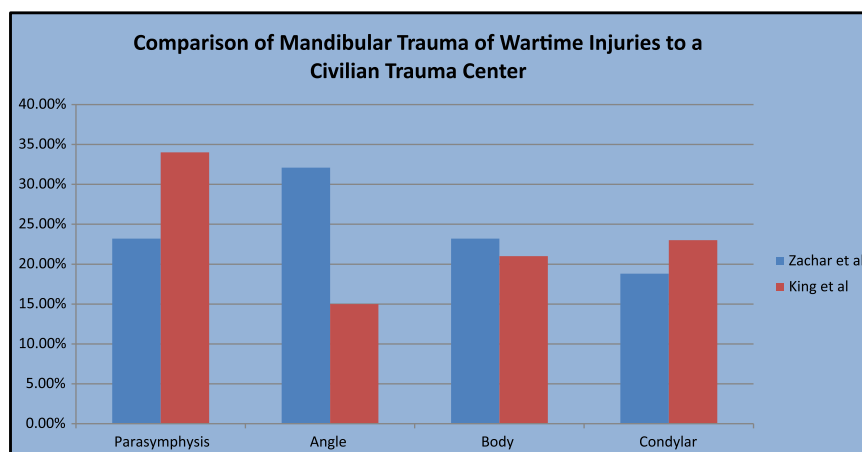


Fig. 6 Comparison of mandibular trauma of combat-related injuries (blue) with those in a civilian trauma center (red).



Fig. 7 Anatomy of the mandible: Fractures are named according to the portion of the mandible through which they pass. From medial to lateral: symphyseal, parasymphysal, body, angle, ramus, subcondylar, condylar, coronoid process above angle/ramus. (From Follmar KE, Baccarani A, Das RR, et al. A clinically applicable reporting system for the diagnosis of facial fractures. *Int J Oral Maxillofac Surg* 2007;36(7):593–600; with permission.)

motion, and vestibular or sublingual ecchymosis. Traction on the anterior mandible will elicit pain in the fracture sites. If the patient is conscious, a neurologic examination will find sensory deficits or motor deficits when there is injury or interruption to the trigeminal or facial nerves.

Simple mandibular fractures can be imaged by the panoramic radiograph. Plain films are of limited value, as images are frequently superimposed and the condyles are difficult to view. When teeth or teeth fragments are unaccounted for, chest x-ray and KUB (an x-ray of the kidneys, ureter, and bladder) films should be taken to rule out aspiration. The computed tomography (CT) scan is invaluable in evaluating condylar fractures and complex mandibular fractures. Three-dimensional reconstruction and stereolithographic models are especially helpful in injuries in which hard tissue is missing or grossly displaced. A comprehensive plan to restore occlusion and continuity of the mandible may include the fabrication of lingual or occlusal splints for use intraoperatively (Box 1, Figs. 8 and 9).

Management

There are 3 basic types of treatment for mandibular fractures: closed reduction, open reduction with internal fixation, and external fixation (Box 2).

Nondisplaced mandibular fractures without occlusal disturbances can be treated with a nonchewing diet. When occlusal disturbances are present and a fracture is minimally displaced, treatment can be the application of intermaxillary fixation for a period of 2 to 3 weeks, depending on the patient's age, health, and fracture type. Displaced fractures generally require open reduction with internal fixation using titanium screws and plates. Because of the high infection rate of open fractures, these should be treated with antibiotic prophylaxis. General anesthesia and paralytics are useful in



Fig. 8 Gunshot wound to mandible. Despite minor external tissue injury, there is extensive comminution of the mandibular body.

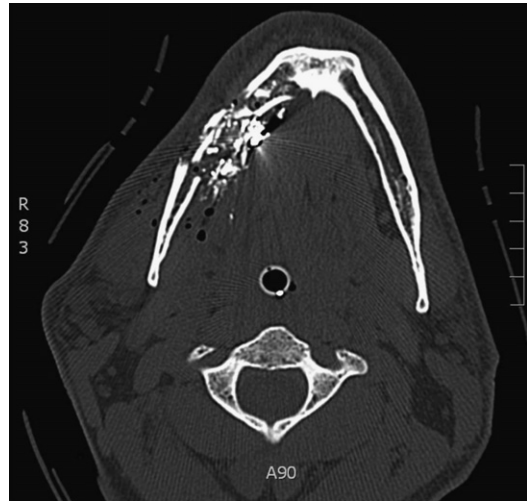


Fig. 9 Axial CT shows extensive fragmentation of right mandibular body from patient in Fig. 8.

Box 1. Clinical indicators of mandibular fracture

- Occlusal changes
- Abnormal opening/deviation
- Anesthesia/paresthesia/dyesthesia
- Vestibular or floor of mouth ecchymoses
- Facial asymmetry
- Loose or fractured teeth

Box 2. Goals of mandibular fracture treatment

- Restore facial contours
- Restore arch form
- Restore occlusion
- Restore function

Table 1 Techniques for closed reduction

Technique for Closed Reduction	Advantage	Disadvantage
Arch bars	Ability to reduce several segments at once, multiple areas to wire into IMF	Time consuming, potential for skin puncture, difficult to remove
Orthodontic brackets	Saves time in operating room, patient comfort	Debond easily, requires orthodontist appointment
Ivy loops	Speed in application, useful for minimally displaced favorable fractures	Less useful with multiple fractures, less control of individual segments
Intermaxillary fixation screws	Speed in application, ease in removal	Cost, potential damage to tooth roots, screws may loosen

reduction, as this will minimize the muscle pull on unfavorable fractures.

Indications for closed reduction of mandibular fractures

- Nondisplaced favorable fractures³
- Grossly comminuted fractures
- Fractures with avulsed tissue—Devascularized bone has limited ability for healing. Placement of plates and screws may further strip the blood supply of these fragments. If possible, flaps should be rotated to improve blood supply to large segments of exposed bone.
- Fractures in children with developing dentitions—Avoiding damage to the developing teeth is key. If placement of arch bars is impossible, consideration should be given to a lingual splint and skeletal fixation with circummandibular and piriform wires.
- Coronoid process fractures
- Condylar fractures—Closed reduction is useful when the occlusion can be reduced and the fracture is minimally displaced.

Treatment with closed reduction

Multiple options are available for reducing the teeth into occlusion via intermaxillary fixation. The most common methods are listed in [Table 1](#).

Indications for open reduction of mandibular fractures

- Displaced unfavorable fractures of the body or parasymphysis³
- Multiple fractures including the midface
- Bilateral condylar fractures
- Edentulous mandible fractures
- Edentulous maxilla with mandible fracture
- When intermaxillary fixation is contraindicated—Open reduction and internal fixation should be considered the preferred treatment in patients with poorly controlled seizures, severe psychiatric or mental impairment, respiratory disorders, or severe nutritional disorders.

Indications for external fixation

- Grossly comminuted fractures—external fixation allows the stabilization and gross approximation of the mandibular segments without compromising the blood supply of small and large bone fragments.

Surgical approach

- Dictated by location and degree of displacement, condition of bony fragments
- Body, angle, and symphysis can usually be plated through vestibular incisions
- Consider extra-oral approach for significantly displaced fractures ([Table 2](#)).

Special considerations for complex open fractures

- Small, devitalized fragments of bone should be removed¹⁴
- Larger fragments should be reduced and fixated
- Use intermaxillary fixation (IMF) to align dentoalveolar fragments
- Cover exposed bone when possible
- Delayed grafting with a healthy, infection-free tissue bed if necessary
- Consider osseous free flap for defect greater than 6 cm
- Open fractures of the mandibular body—this area is exceptionally difficult to treat. Comminution and significant displacement frequently interrupt the centripetal blood supply of the inferior alveolar vessels and make this area especially prone to infection and necrosis ([Figs. 10–17](#)).

Table 2 Surgical approaches to mandibular fractures

Surgical Approach	Region Accessed	Benefits	Complications
Submandibular	Body/Angle	Excellent visualization of inferior border	Scarring, potential facial nerve injury
Preauricular	Temporomandibular joint	Ability to fixate condylar head, restore vertical height of posterior mandible	Scarring, potential facial nerve injury
Retromandibular	Neck of condyle	Ability to fixate subcondylar fracture, restore vertical height of posterior mandible	Scarring, potential facial nerve injury
Vestibular/ intraoral	Symphysis, parasymphysis, body, and angle	No facial scarring, avoids facial nerve	Difficult to visualize inferior border and lingual plate



Fig. 10 Initial treatment of this patient included debridement of devitalized bony fragments, stabilization of teeth in arch bars, and reapproximation of tissues to cover exposed bone.



Fig. 11 Patient after initial debridement and soft tissue reapproximation.



Fig. 12 To preserve blood supply to the mandible, an external fixator is applied to stabilize the bony fragments.



Fig. 13 Integra matrix wound dressing is applied to provide scaffold for capillary growth and support of split thickness skin graft.



Fig. 14 Wound healing after maturation of skin graft.

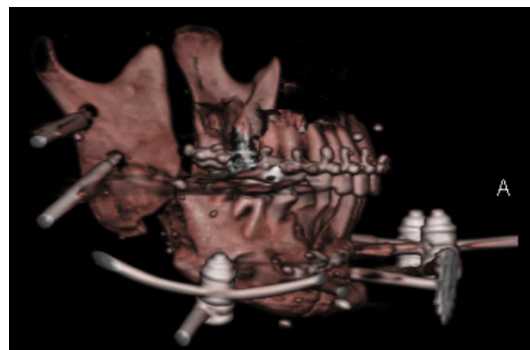


Fig. 15 After initial bone healing, a large defect of the mandibular body remains with minimal bony union.

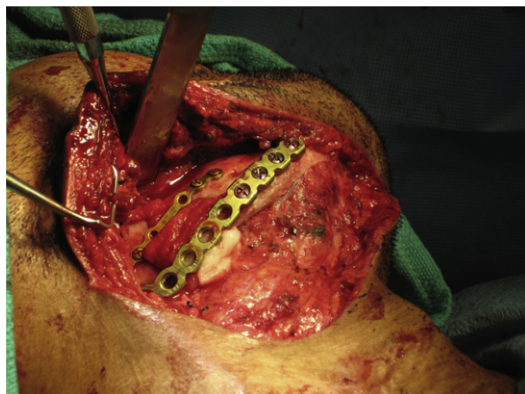


Fig. 16 External fixator is removed and mandible reconstructed with Bone morphogenetic protein.

Complications

Complications of mandibular fractures are fairly common, with a wide range of infection rates reported (between 4% and 50%).¹⁵ These complications include infection, osteomyelitis, malunion, nonunion, and nerve disturbances. Contributing factors to complications include teeth in the line of fracture, antibiotic use, compliance of patient, and substance abuse.^{16,17} In a prospective study, Chole and Yee¹⁸ found that prophylactic antibiotic use is shown to reduce the risk of infections in facial fractures from 42.2% to 8.9%.

Avulsive, comminuted wounds, or those with diminished blood supply, should be considered separately. Fractures in which the central blood supply of the mandible has been interrupted are particularly troublesome and prone to resorption, nonunion, and necrosis. A prolonged course of antibiotic therapy is indicated in these especially infection-prone patients.^{19,20}

- **Infection**—the most commonly encountered complication of mandibular fractures, especially in complex fractures. Infections in mandibular fractures are generally polymicrobial and are more common when teeth are involved in the line of fracture.¹⁶ Incision and drainage should be performed if the infection is localized to the surgical area. Rigid fixation should be maintained for 4–6 weeks, at which point the hardware can be removed. If the infection involves loose bony fragments or hardware, they should be removed until bleeding bone can be visualized. Rigid

fixation should be applied through a reconstruction plate or external fixator.

- **Nonunion** occurs when a fracture fails to heal within 6 months. This is caused by infection or mobility at the fracture site.
- **Malunions** occur when the bone heals, but malocclusion results. Orthodontics should be considered for minor occlusal changes. A full orthognathic surgery workup is indicated for major occlusal discrepancies.
- **Nerve injury** to the inferior alveolar nerve or mental nerves is common. Less commonly, the facial nerve can be injured during extra-oral access to fractures. Nerve injuries should be monitored for resolution. These patients should be treated medically if they develop dyesthesia and referred to a specialist if their symptoms do not improve.

Summary

Fractures of the mandible are among the most common facial injuries. Invasiveness of treatment should be determined by the extent of injury: degree of displacement, number of fractures, the patient's health status, and concomitant injuries. Complex, comminuted, and avulsive injuries frequently seen in combat will require coordination with multiple specialties to provide the best treatment. Stabilization treatment with arch bars or external fixators and splints is often desirable when fractures are highly comminuted or the soft tissue envelope is compromised by tissue loss or burns. In severe injuries, many times reconstruction will take several surgeries. Debridement of necrotic tissue and devascularized bone and skin grafting often are necessary before reconstruction. Microvascular or myocutaneous flaps should be considered with significant tissue loss and osteocutaneous flaps when large continuity defects are present.

Most mandible fractures are repaired in a single operation. Those caused by explosives and high-velocity projectiles are more complex. Research should continue to focus on improving outcomes for these patients. Advances in tissue engineering, bone regeneration, and composite tissue allografting will have to continue if we hope to restore facial form and function for our combat wounded.

References

1. Haskell BS, Arm R, Stroop 3rd JR, et al. The role of the dentist in archaeologic investigation: an unusual facial fracture with healing occurring 3,000 years ago. *Quintessence Int* 1985;16(1):95–101.
2. Widell T, Chief editor: Kulkarni R. Mandible fracture in emergency medicine. Available at: <http://emedicine.medscape.com/article/825663-overview>. Accessed September 20, 2012.
3. Fonseca RJ, Walker, Betts. *Oral and maxillofacial trauma*. 3rd edition. St Louis (MO): Saunders; 2004. p. 479–522. ISBN:9780721601830.
4. Brooks SM. Thomas Brian Gunning, D.D.S. and—the day Seward was stabbed. *TIC* 1985;44(4):7–10.
5. Mukerji R, Mukerji G, McGurk M. Mandibular fractures: historical perspective. *Br J Oral Maxillofac Surg* 2006;44(3):222–8.
6. Ellis E. Treatment methods for fractures of the mandibular angle. *Int J Oral Maxillofac Surg* 1999;28(4):243–52.
7. Madsen MJ, McDaniel CA, Haug RH. A biomechanical evaluation of plating techniques used for reconstructing mandibular symphysis/parasymphysis fractures. *J Oral Maxillofac Surg* 2008;66(10):2012–9.
8. Lee HB, Oh JS, Kim SG, et al. Comparison of titanium and biodegradable miniplates for fixation of mandibular fractures. *J Oral Maxillofac Surg* 2010;68(9):2065–9.

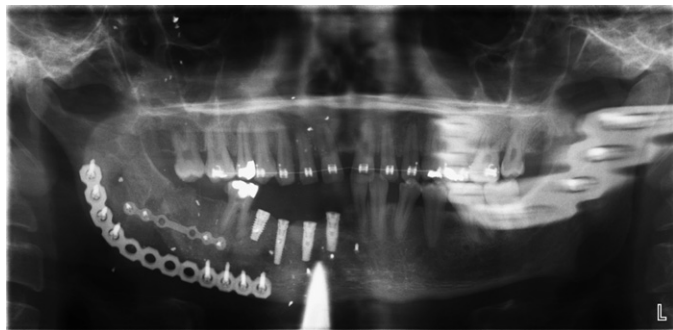


Fig. 17 Final reconstruction with dental implants.

9. Wald Jr RM, Abemayor E, Zemlenyi J, et al. The transoral treatment of mandibular fractures using noncompression miniplates: a prospective study. *Ann Plast Surg* 1988;20(5): 409–13.
10. Lew TA, Walker JA, Wenke JC, et al. Characterization of cranio-maxillofacial battle injuries sustained by United States service members in the current conflicts of Iraq and Afghanistan. *J Oral Maxillofac Surg* 2010;68(1):3–7.
11. King RE, Scianna JM, Petruzzelli GJ. Mandible fracture patterns: a suburban trauma center experience. *Am J Otolaryngol* 2004; 25(5):301–7.
12. Dorland WAN. *Dorland's illustrated medical Dictionary*. 30th edition. Philadelphia: WB Saunders; 2003.
13. Shetty V, Atchison K, Der-Matrosian C, et al. The mandible injury severity score: development and validity. *J Oral Maxillofac Surg* 2007;65(4):663–70.
14. Hale RG, Hayes DK, Orloff G, et al. *Combat casualty care: lessons learned from OEF & OIF: maxillofacial and neck trauma [DVD]*. Los Angeles (CA): Pelagique, LLC; 2010.
15. Chan DM, Demuth RJ, Miller SH, et al. Management of mandibular fractures in unreliable patient populations. *Ann Plast Surg* 1984; 13:298.
16. Ellis E. Outcome of patients with teeth in the line of mandibular angle fractures treated with stable internal fixation. *J Oral Maxillofac Surg* 1996;44:858.
17. Serena-Gomez E, Passeri LA. Complications of mandible fractures related to substance abuse. *J Oral Maxillofac Surg* 2008;66(10): 2028–34.
18. Chole RA, Yee J. Antibiotic prophylaxis for facial fractures. A prospective, randomized clinical trial. Antibiotic prophylaxis for facial fractures. A prospective, randomized clinical trial. *Arch Otolaryngol Head Neck Surg* 1987;113(10):1055–7.
19. Kyle P, Hayes D, Blice J, et al. Prevention and Management of infections associated with combat-related Head and Neck injuries. *J Trauma* 2008;64:S265–76.
20. Kyzas PA. Use of antibiotics in the treatment of mandible fractures: a systematic review [review]. *J Oral Maxillofac Surg* 2011;69(4): 1129–45.

Management of Midface Maxillofacial Trauma

Michael A. Gentile, DMD ^{a,b,c,*}, Andrew J. Tellington, DDS ^{a,b}, William J. Burke, DMD ^{a,b}, Michael S. Jaskolka, MD, DDS ^{d,e,f}

KEYWORDS

- Midface fractures • LeFort • Zygomaticomaxillary complex (ZMC) • Orbital • Nasal-orbital-ethmoid (NOE)
- Frontal sinus • Cranialization • Obliteration

KEY POINTS

- The maxilla, palate, zygomaticomaxillary complex, nasal bones, orbits, nasal-orbital-ethmoid complex, and frontal sinus may be affected by midface trauma.
- Forces directed onto the midfacial skeleton are absorbed and transmitted through vertical and horizontal buttresses.
- By reconstructing and stabilizing the vertical and horizontal buttresses of the midface, occlusal forces can be tolerated and facial height, width, and projection can be restored.
- Complications of midface trauma include bleeding, malunion/nonunion, neurologic complications, ocular complications, and complications involving the lacrimal system.
- Frontal sinus fractures can be followed with close observation or treated surgically with anterior table reconstruction alone or in combination with sinus obliteration or cranialization.
- The decision to treat a frontal sinus fracture is dependent on the amount of bony displacement, the involvement of the posterior table and intracranial contents, and the condition of the nasofrontal outflow tract.

Introduction

The management and surgical treatment of midface maxillofacial trauma can present one of the most challenging undertakings for the maxillofacial surgeon. The midfacial skeleton and its soft tissue attachments protect the brain and eyes from injury and are closely related to the senses of vision and smell. Speech, mastication, and facial appearance can all be affected by midfacial trauma. Accurate correction of the bony skeleton to the preinjury state is vital to the restoration of function and esthetics.

Disclaimer: The views expressed in this article are those of the authors and do not reflect the official policy of the Department of Navy, Department of Defense, or U.S. Government.

^a Department of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, MD 20889, USA

^b Oral and Maxillofacial Surgery Residency Program, National Capital Consortium, 4301 Jones Bridge Road, Bethesda, MD 20814, USA

^c Department of Surgery, Uniformed Services University of Health Sciences, 4301 Jones Bridge Road, Bethesda, MD 20814, USA

^d First Appalachian Craniofacial Deformity Specialists, Multi-Disciplinary Cleft and Craniofacial Disorders Clinic, Women and Children's Hospital, Charleston Area Medical Center, 830 Pennsylvania Avenue, Suite 302, Charleston, WV 25302, USA

^e Charleston Division, Department of Surgery, West Virginia University, 3110 MacCorkle Avenue SE, Charleston, WV 25304, USA

^f Department of Oral and Maxillofacial Surgery, University of North Carolina, 101 Manning Drive, Chapel Hill, NC 27514, USA

* Corresponding author. Department of Oral and Maxillofacial Surgery, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, MD 20889.

E-mail address: michael.a.gentile@med.navy.mil

Initial assessment

The initial evaluation and management of midface trauma should be directed at stabilization of the patient. Motor vehicle collisions represent the most common cause of facial trauma. Other causes include assaults, falls, sporting injuries, and home and occupational accidents. The high-energy nature of these injuries often leads to multisystem involvement and, therefore, a thorough, systematic evaluation of the entire patient should precede the management of their facial injury. The most common concomitant injuries in patients with pan-facial fractures include intracranial injury or hemorrhage, abdominal organ injury, pneumothorax, pulmonary contusion, spine fracture, rib or sternum fracture, extremity fracture, and pelvic fracture. Trauma centers are especially equipped to deal with the evaluation and management of these injuries. Specially designed teams directed by trauma surgeons and emergency room physicians lead the initial management of these patients. Oral and maxillofacial surgeons are most commonly consulted after the initial evaluation and management of the most life-threatening injuries.

Of particular concern in patients with midface trauma are:

- The cervical spine
- The airway
- Hemorrhage

Midface fractures are positive predictors of cervical spine fractures and dislocations. The cervical spine should be immobilized until cervical fractures have been ruled out by imaging or clinical examination. If an injury is identified, head and neck immobilization and positioning during the repair of

the midface injury should be coordinated with the spinal surgeon.

Airway obstruction can lead to asphyxia and death after midfacial trauma. Bleeding, fractured teeth, oral secretions, vomitus, foreign bodies, and edema can affect airway patency. If the airway obstruction cannot be cleared or controlled, a definitive airway should be placed via endotracheal intubation or cricothyrotomy. Oral endotracheal intubation is successful in the hands of experienced emergency physicians using rapid sequence induction. In terms of midface fracture repair, nasal intubation allows for the simplest establishment of the dental occlusion. However, the increased difficulty, decreased speed and possibility of a concomitant basilar skull injury with concern for cranial intubation deter most providers from attempting this in an emergency setting. If needed, nasal intubation is better performed as a non-emergent procedure in the operating room prior to fracture repair. When attempts at oral intubation are unsuccessful, a surgical airway via cricothyrotomy provides the fastest approach. Cricothyrotomy should then be converted to a tracheostomy, if necessary, in a controlled environment with less risk of losing airway control.

With a secure airway, uncontrolled bleeding can be addressed. The midface has a robust blood supply, with contributions from both the internal and other branches of the external carotid arteries. The sphenopalatine and other branches of the internal maxillary artery can be significantly damaged during midfacial trauma. Although rare, life-threatening hemorrhage can result. When severe epistaxis is encountered, direct pressure via anterior and posterior nasal packs can be used. Often, posterior bleeding is encountered with drainage into the nasopharynx. A 10-French Foley catheter can be inserted through each nare, inflated with sterile water, and then pulled anteriorly. This procedure tamponades off the posterior chamber and in combination with an anterior packing provides a simple way of nasal packing. Anterior nasal packs can be accomplished with the layering of ribbon gauze or with the use of expandable sponges such as Merocel (Medtronic, Inc, Mystic, CT, USA) or Rhino Rocket (Shippert Medical Technologies Corporation, Centennial, CO, USA). Bleeding from intraoral wounds can be controlled with gauze packing, suturing, or electrocautery. If the hemorrhage has caused significant volume loss, fluid should be replaced with lactated Ringer solution or normal saline to restore blood pressure until blood can be typed and crossmatched. Alternatively, the patient can be transfused with O-negative blood. Once the primary advanced trauma life support survey has been completed and the patient's airway and cardiopulmonary status have been stabilized, the secondary survey, including a more detailed facial examination, can be accomplished.

Clinical examination

The awake patient can be questioned about their occlusion, sensory changes, and pain. The patient's subjective assessment of their bite can be one of the most sensitive measures when evaluating for the presence of a maxillary or mandibular fracture. Edema within the temporomandibular joint may also cause changes in occlusion and should be taken into consideration. Paresthesia and numbness of the upper lip, side of the nose, and maxillary gingiva suggest a fracture involving the infraorbital nerve and are common with maxillary and orbital fractures. Pain is also a common finding in the region of a fracture.

The physical examination is best accomplished when performed systematically. We prefer a top-down/outside-in approach. The soft tissue is first inspected for lacerations, edema,

and ecchymosis. Abnormalities and asymmetries in midfacial height, width, and projection are assessed. The intercanthal distance is measured, and epiphora and rhinorrhea are noted if present. A cranial nerve examination is performed, including a detailed assessment of visual acuity and extraocular movements. Bimanual palpation can then be undertaken to assess for bony steps, mobility, crepitus, and tenderness. Palpation begins with the frontal bones and supraorbital rims. It then extends to the lateral orbits, zygomatic arches, and zygomas. The infraorbital rims are next addressed, followed by the medial orbits and nasal bones. The maxilla is then palpated. In addition to palpation, the maxilla can be grasped around the anterior maxillary teeth, with the thumb inferior to the anterior nasal spine and the forefinger at the depth of the palatal vault to assess for mobility. An intraoral examination is accomplished. The intraoral soft tissues are inspected for lacerations and ecchymosis. The teeth should be examined for fractures, luxations, and avulsions. The alveolar bone is assessed for displacement and mobility. Maximum incisal opening, lateral excursive movements, and protrusive movements are recorded and the occlusion is assessed.

A radiographic examination should always accompany the clinical examination in the patient with facial trauma. Although plain films can be useful for identification of specific fracture elements, computed tomography (CT) has become the standard for evaluating midfacial injuries. CT scans provide information in 3 planes of space (axial, coronal, and sagittal), and can be used as a standalone radiographic modality. The CT data can also be reconstructed into a three-dimensional (3D) image, increasing its usefulness. At a minimum, we prefer to order a non-contrast maxillofacial CT with 1-mm to 2-mm axial slices and coronal, sagittal, and 3D reconstructions (Fig. 1).

Maxillary and LeFort fractures

Anatomy

The maxilla, palate, nasal bones, and zygomas comprise most of the midfacial skeleton. The ethmoids, greater wing of the sphenoid, and frontal bone comprise elements of the bony orbit and connect the anterior facial skeleton to the cranial base. Forces directed onto the midfacial skeleton are absorbed and transmitted through vertical and horizontal buttresses. These buttresses constitute areas of dense, thick bone that support the maxilla and are more resistant to deformation when forces are applied. They are not only important in protecting the vital structures of the midface but they are also essential landmarks used during reconstruction. The buttresses provide higher-quality bone for internal fixation and guide reconstruction of facial height, width, and projection.

The midface is more resistant to vertical forces than horizontal and shear forces. This resistance is because of the strength of the 4 vertical buttresses (Fig. 2):

- Nasomaxillary (medial)
- Zygomaticomaxillary (lateral)
- Pterygomaxillary (posterior)
- Ethmoid-vomerian or septal (midline)

The paired nasomaxillary buttresses extend from the frontal bone to the nasal bones and medial orbit, and along the pyriform apertures and end at the maxillary alveolus in the region of the maxillary canines. Laterally, the pterygomaxillary

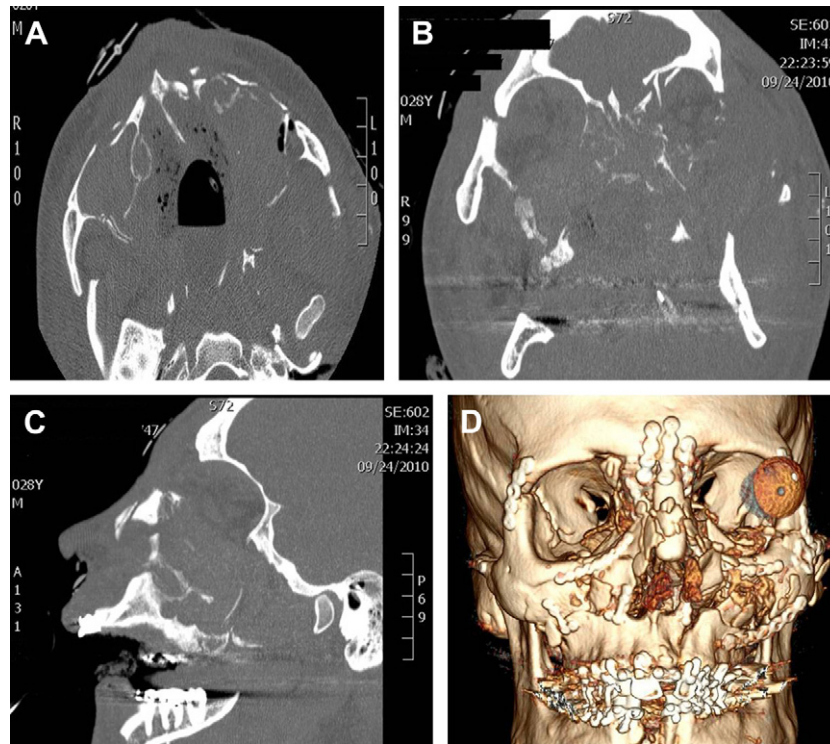


Fig. 1 CT scan of comminuted midface fracture. (A) Axial slice, (B) coronal slice. (C) sagittal slice, and (D) 3D reconstruction after surgical repair.

buttresses extend from the frontal bone, down the lateral orbital rims to the zygoma and end at the maxillary alveolus in the region of the maxillary second molars. The pterygomaxillary buttresses provide posterolateral support and extend from the pterygoid plates of the sphenoid bone to the posterior maxilla. The midline ethmoid-vomerian or septal strut joins the frontal bone and cranial base with the midpalatal suture.

Although the horizontal buttresses have less impact on force dissipation, they are important for the restoration of facial width. The horizontal buttresses include (Fig. 3):

- Superior orbital rims (superior)
- Inferior orbital rims/zygomatic arch (central)
- Maxillary alveolus (inferior)

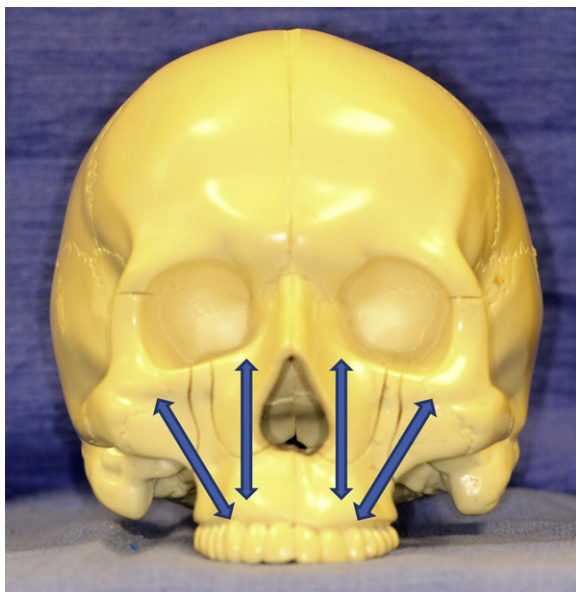


Fig. 2 The vertical buttresses of the midface. Arrows indicate the nasomaxillary buttresses medially and the zygomaticomaxillary buttresses laterally.

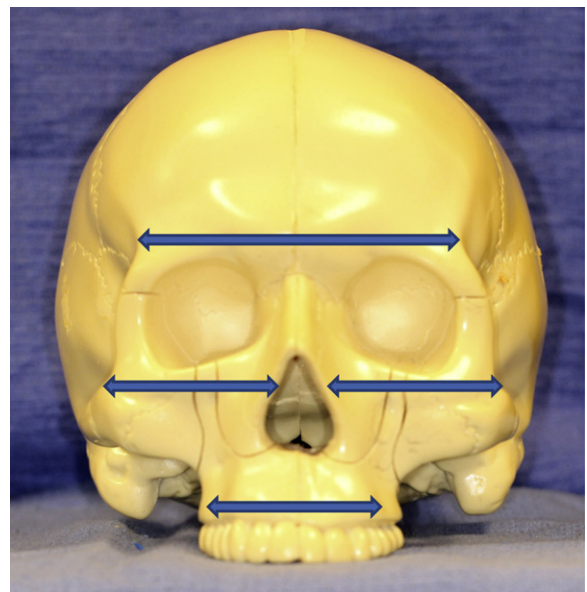


Fig. 3 The horizontal buttresses of the midface. Arrows indicate the superior orbital rims (*superior*), the inferior orbital rims/zygomatic arches (*central*), and maxillary alveolus (*inferior*).

Superiorly, the frontal bone extends from 1 superior orbital rim to the other, bridged by the bone at the nasofrontal region. The central aspect of the midface is composed of a horizontal buttress extending from 1 zygomatic arch and inferior orbital rim, across the midline and pyriform aperture, to the contralateral counterpart. The most inferior horizontal midface buttress is the maxillary alveolus.

Classification

The work of René LeFort has stood the test of time in the classification of maxillary and midfacial bony trauma. LeFort was a French army surgeon who conducted a series of experiments in Lille, France at the turn of the twentieth century. He published his work, "Etude expérimentale sur les fractures de la machoire supérieure" in 1901. LeFort took whole human cadavers and severed heads and inflicted traumatic forces on the midface with variations in force and vector. He then boiled the heads to help remove the skin and examined the skulls. LeFort found that most fractures occurred along 3 "great lines of weakness," which are now referred to as LeFort I, II, and III fractures.

A LeFort I fracture, or Guerin fracture, is a transverse fracture that occurs along the maxilla (Fig. 4A). These fractures typically result from a force directed above the level of

the teeth. They extend from the pyriform rims, through the anterior, lateral, and posterior walls of the maxillary sinus, and through the pterygoid plates of the sphenoid bone. The nasal septum is often fractured. Because of the pull of the medial and lateral pterygoid muscles, these fractures often result in a posterior and inferior positioning of the posterior maxilla, resulting in an anterior open bite.

A LeFort II fracture is a pyramidal fracture that extends from the maxillary tuberosity through the medial aspect of the inferior orbital rim in the region of the zygomaticomaxillary suture, through the lacrimal bone, and up to the nasofrontal suture (see Fig. 4B). Again, the nasal septum is often fractured, and the nasal bones may be displaced. A force directed at the nasal bones is responsible for this fracture pattern.

A LeFort III fracture includes the zygomas and a portion of the lateral and inferior orbit (see Fig. 4C). These fractures are caused by forces directed at the level of the orbits. They extend through the zygomaticofrontal and zygomaticotemporal sutures, course along the lateral orbit, through the inferior orbital fissure and medial orbit to the nasofrontal suture. Posteriorly, they end at the pterygomaxillary junction. LeFort III fractures represent a true craniofacial disjunction, that is, the separation of the midfacial skeleton from the cranial base.

Although useful in organizing and communicating the nature of bony injuries, the LeFort classification scheme does not always represent the fracture pattern seen in patients.

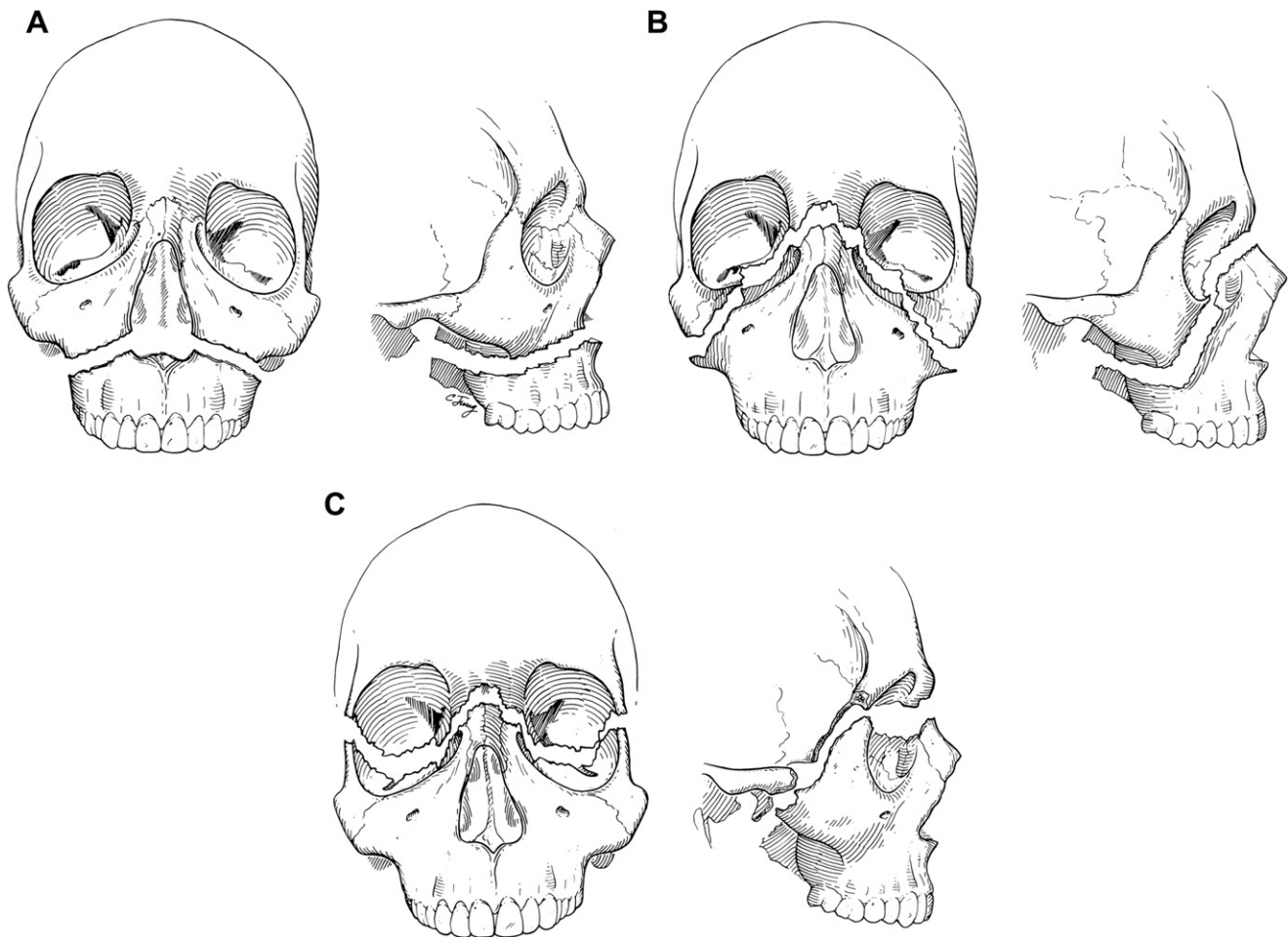


Fig. 4 The LeFort classification of midface fractures: (A) LeFort I, (B) LeFort II, and (C) LeFort III. (From Salin MB, Smith BM. Diagnosis and treatment of midface fractures. In: Fonseca RJ, editor. Oral and maxillofacial trauma. vol. 2. St Louis (MO): Elsevier; 2005. p. 645–6; with permission.)

Fractures are dependent on the position, vector, and intensity of the force directed at the bony skeleton. They are rarely completely symmetric bilaterally and often occur unilaterally. Fractures may also involve the palate, which is discussed later. It may be more accurate to describe and identify the specific fracture elements present when planning and treating midface fractures.

Diagnosis and treatment planning

The general approach to the management of midface trauma was discussed earlier. Maxillary and LeFort fractures may present with a variety of clinical findings (Fig. 5). Identification of these common findings is helpful with diagnosis and treatment planning. Soft tissue lacerations, if present, likely indicate an area of direct force. Edema may alert the clinician to the region of likely fractures, but may also obscure any change in facial width or projection. Nasal bleeding is common to midface fractures because of the disruption of the nasal septum and mucosa. Pain, ecchymosis, and bony steps along the fractures lines may be appreciable on visual inspection and palpation. In addition, a change in occlusion is often found.

For LeFort I fractures, maxillary mobility may be present. However, the absence of mobility does not preclude a fracture. If the maxilla has been impacted, there may be no mobility and the anterior facial height may be decreased. There is typically an anterior open bite caused by the posterior and inferior force placed on the maxilla by the medial and lateral pterygoid muscles. Ecchymosis in the region of the greater palatine

foramen, or Guerin sign, and ecchymosis in the buccal vestibule also indicate a LeFort I fracture.

LeFort II fractures typically present with periorbital and subconjunctival ecchymosis in addition to the findings outlined earlier. A bony step at the infraorbital rim may also be detectable. Disruption of the infraorbital nerve leads to anesthesia of the upper teeth, gingiva, upper lip, and lateral aspects of the nose. If the orbital floor has been disrupted with entrapment of the inferior rectus, diplopia with restricted superior gaze is present. If the nasal bones and maxilla are mobile, a LeFort II fracture should be suspected. A dish-faced appearance may be present as a result of decreased nasal projection. In addition, rhinorrhea of the cerebrospinal fluid (CSF) may be present, suggesting a basilar skull fracture with involvement of the dura.

LeFort III fractures involve mobility of the maxilla, nasal bones, and zygomas as a single unit. A palpable bony step may be present at the zygomaticotemporal or zygomaticofrontal suture. As with LeFort II fractures, bilateral periorbital edema (raccoon eyes) and CSF rhinorrhea may be present. Lengthening of the facial height, orbital hooding, and enophthalmos are also typically present. Ecchymosis over the mastoid region (Battle sign) may be present, in addition to CSF otorrhea and hemotympanum.

Features of the clinical examination should be supported by the radiographic examination. A CT scan of the head and maxillofacial region detects fracture lines, bony displacement, air-fluid levels in the paranasal sinuses, orbital entrapment, and intracranial involvement. They provide exquisite detail and have revolutionized the diagnosis of specific fracture entities.

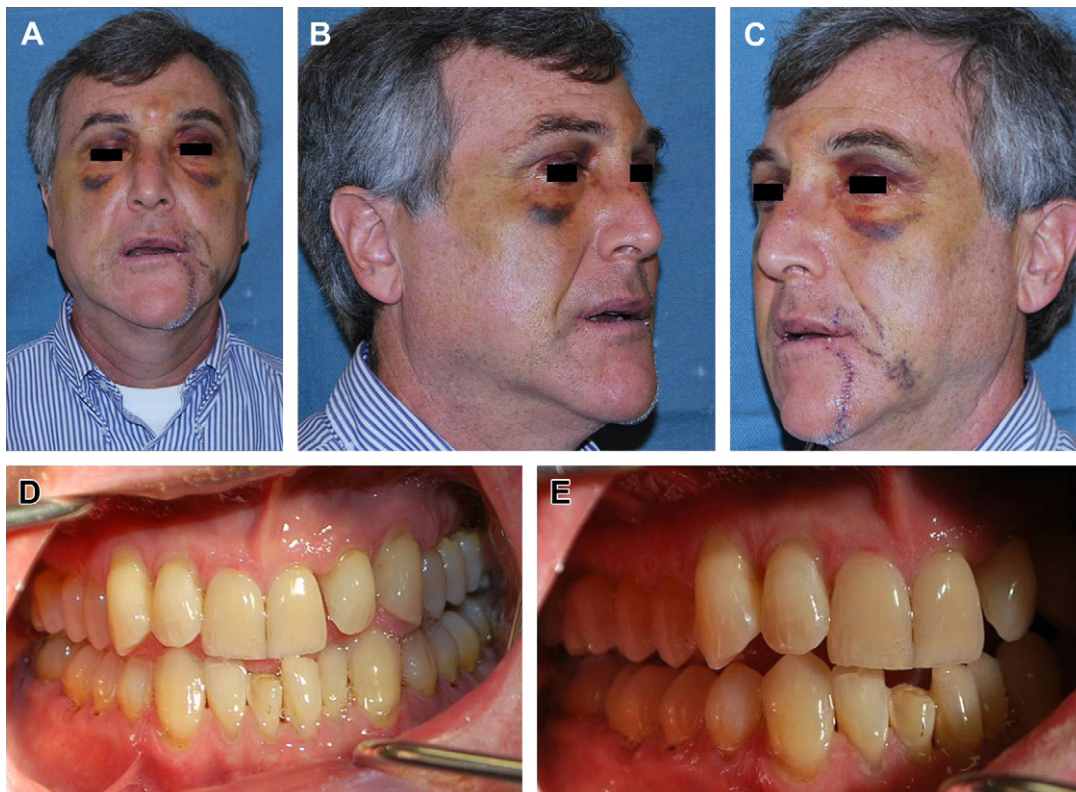


Fig. 5 Typical clinical presentation after midface trauma. Patient sustained LeFort II level fracture after fall with blunt force trauma. Clinical photographs represent patient presentation 1 week after trauma before surgical repair. (A) Frontal view showing resolving edema with bilateral periorbital ecchymosis and deviation of nasal dorsum. (B, C) Three-quarter view showing midface flattening. (D, E) Occlusal views showing class III malocclusion with edge-to-edge incisor relationship.

Surgical treatment and postoperative management

The goals of treatment of midface fractures are to restore function and esthetics. In terms of function, reestablishment of the preinjury dental occlusion is essential. By reconstructing and stabilizing the vertical and horizontal buttresses of the midface, occlusal forces can be tolerated and facial height, width, and projection can be restored.

The timing of repair for maxillary and midfacial fractures is often a source of debate. Although early repair may facilitate reduction, the patient's systemic status may prevent surgical correction in the first few days after the injury. It is therefore more common to complete the definitive fracture reduction and fixation after the patient has been stabilized, a definitive airway (if necessary) has been placed, and the patient's edema has begun to resolve. This strategy gives the maxillofacial surgeon time to review all of the relevant clinical and radiographic data and plan surgery that best treats the patient's needs. We believe the ideal timing of surgery to be between 7 and 10 days after trauma. At this point, the bulk of edema has resolved and the fractures are still simple to mobilize and reduce. Beyond 10 days, reduction may become more difficult because of osseous healing and fibrosis. There are obviously exceptions to this rule, such as orbital entrapment and hemorrhage control. The timing of surgery is patient-specific and must take all factors into account.

A stable airway must be secured for repair of midface trauma. For most LeFort I fracture cases, a nasoendotracheal tube can be placed by the anesthesiologist, with fiber-optic guidance, if necessary. A nasoendotracheal tube may interfere with LeFort II and III level fractures because of the involvement of the nose. Also, in cases involving basilar skull fractures, there may be hesitancy to place a nasal tube for fear of intracranial intubation. In these cases, an oral endotracheal tube can be placed behind the existing dentition or through dental gaps. If this procedure is not possible, a submental intubation technique can be used. It is important that the patient's final occlusion can be established without interference from the endotracheal tube. In certain cases, a tracheostomy may be indicated. Examples of these cases include patients with cervical spine injuries and patients requiring prolonged ventilatory support.

In cases involving fractures with minimal displacement and no change in occlusion, no treatment other than a nonchew diet for approximately 4 weeks may be indicated. However, these cases are rare. The ideal repair of maxillary and LeFort level fractures involves open reduction. When open reduction is not possible, closed reduction techniques involving maxillomandibular fixation (MMF) for a period of 4 to 6 weeks can be used. In cases in which there is displacement of the maxilla with an occlusal discrepancy, Rowe disimpaction forceps can be used to mobilize and reduce the maxilla (Fig. 6). With closed reduction, facial height, width, and projection are based off the dental occlusion. Because there is no direct visualization of the fractures, it is difficult to control the vertical position of the maxilla, and autorotation of the maxillomandibular unit may lead to a change in facial height and projection. The severely comminuted midface injury may also create a challenge for open reduction. In patients who cannot undergo autogenous grafting or for those who do not tolerate MMF because of medical or psychological reasons, external fixation with a halo head frame can be used.



Fig. 6 Rowe disimpaction forceps are used to mobilize and reduce the maxilla in a patient with a LeFort I level fracture.

Despite these exceptions, the most common treatment of maxillary and LeFort fractures remains open reduction with internal fixation. The general treatment approach involves:

- Exposure of the fractures
- Mobilization and reduction of fracture segments
- Establishment of occlusal relationships (MMF)
- Rigid or semirigid fixation
- Bone grafting when necessary
- Resuspension/repair of soft tissue injuries

Our preference is to place Erich arch bars before the surgical incision. Any modality for MMF can be used if a stable occlusion can be attained. It is our experience that arch bars provide the most stability and ease of reduction when placing patients into MMF. They also provide the most control over the occlusion in the postoperative period.

The most common approach for exposure of a LeFort I level fracture is the buccal vestibular incision (Fig. 7). This incision extends from 1 zygomaticomaxillary buttress to the other at a level approximately 5 mm superior to the mucogingival junction. Excellent exposure of the nasomaxillary buttresses, zygomaticomaxillary buttresses, and the anterior and lateral walls of the maxillary sinus is attained. Dissection up to the inferior orbital rims is possible through this approach, if necessary. Once exposed, the fractures are mobilized using Rowe forceps. Alternatively, an instrument such as a Tessier

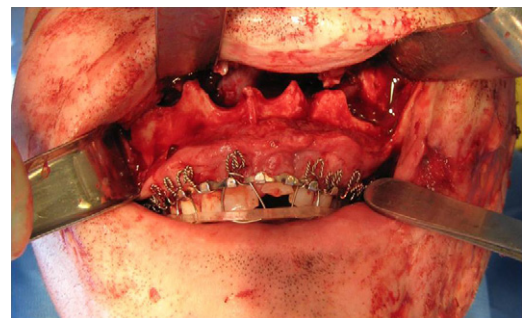


Fig. 7 Standard exposure of LeFort I fracture by buccal vestibular incision.

mobilizer can be placed behind the maxillary tuberosity to apply anterior and medial force to the impacted maxilla. Once the bony segments are mobilized, the patient is placed into MMF. The maxillomandibular unit is then rotated in the glenoid fossa, making sure that the mandibular condyle is passively seated and the fractures are reduced. Failure to properly seat the condyle before fixation causes a class II malocclusion, with an anterior open bite on the affected side on release from MMF (assuming a class I preinjury occlusion). Next, the fractures are fixated with either bone plates or interosseous wiring. The fixation pattern and method are dependent on the specific fracture location and bone available. The most common method is to use 4-point fixation at the zygomaticomaxillary and nasomaxillary buttresses with 1.5-mm to 2.0-mm miniplates (Fig. 8). If bony gaps exist, autogenous grafting can be used (Fig. 9). The patient is then released from MMF, the occlusion is checked to confirm reproducibility, and the wounds are closed.

Open reduction of LeFort II fractures may require fixation across the infraorbital rim or the nasofrontal region. If possible, 3-point or 4-point fixation is ideal. The buccal vestibular incision described earlier can be used to expose the zygomaticomaxillary buttresses. The decision to expose the infraorbital rim is often dependent on a palpable step or cosmetic deformity in the rim or in cases in which orbital floor exploration and reconstruction is indicated (ie, orbital floor blow-out with enophthalmos or entrapment). The infraorbital rim can be accessed via a transconjunctival, subciliary, infraorbital, or lower lid approach. Each has distinct advantages and disadvantages, which are discussed in a later section. Once the rim is exposed, the fractures are mobilized and reduced as described earlier, and a 1.0-mm to 1.3-mm plate can be placed for fixation. The nasofrontal region can be exposed via an existing laceration, bilateral lynch incision, open-sky approach, or coronal incision. These approaches are discussed later. A low-profile plate (1.3 mm) can then be placed for fixation.

Open reduction of a LeFort III fracture requires exposure of the zygomaticofrontal suture in addition to those discussed earlier. Plates of at least 1.3-mm strength should be placed to fixate this area. The coronal flap provides excellent exposure of this area in addition to the nasofrontal region and is the preferred approach. Alternatively, an upper lid (blepharoplasty) or lateral eyebrow incision can be used to access the zygomaticofrontal suture. This approach, in combination with the approaches to access the nasofrontal region and

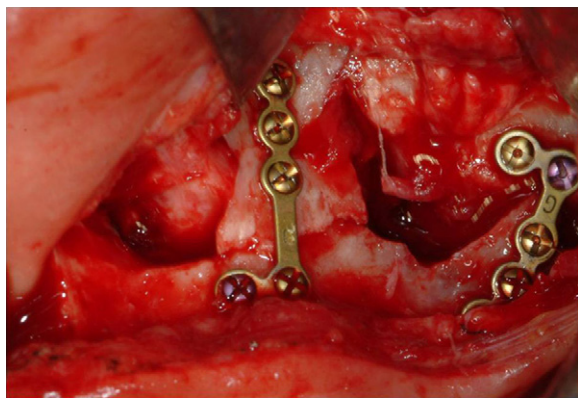


Fig. 8 Fixation of the left maxilla at the LeFort I level with 2.0-mm miniplates. Five-hole L-plates were placed at the nasomaxillary and zygomaticomaxillary buttresses.

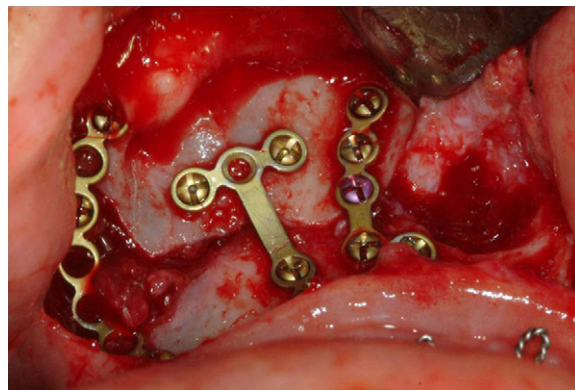


Fig. 9 An autogenous cortical bone graft taken from the mandibular ramus was used to bridge a bony gap in this LeFort I fracture.

zygomaticomaxillary buttresses, can expose all of the areas needed for fixation. We prefer to sequence fracture fixation from top-down and outside-in.

When extensive subperiosteal dissection is performed in the midface, ptosis of the midfacial soft tissue may result if it is not resuspended. Midface periosteum can be resuspended to the orbital rim and the lateral and temporal soft tissue can be resuspended to the temporal fascia or temporal or parietal bone.

Postoperatively, patients are kept on a nonchew diet for 4 to 6 weeks. The patient should avoid any occlusal force that could cause micromovement across the fracture lines. The arch bars are kept in place during this period and light guiding elastics can be used to control the occlusion. There is controversy over whether antibiotics should be used in the postoperative period. Some advocate only a single preoperative dose of antibiotics, unless there is an existing infection or if the inciting trauma caused a contaminated wound. Others have promoted the use of postoperative antibiotics for 7 to 14 days. We prefer a 7-day course of antibiotics, with broad spectrum coverage to include oral and sinus flora (ampicillin and sulbactam, amoxicillin and clavulanate). Patients can also be placed on a decongestant and oxymetazoline nasal spray to encourage drainage of the maxillary sinus. Patients should be counseled about sinus precautions, proper nutrition, and limitation of physical activity. At the end of the 4-week to 6-week period, the stability of the maxilla is assessed. If stable, the arch bars are removed and the patient's diet and activity level are slowly advanced.

Palatal fractures

Classification

Palatal fractures occur in approximately 8% of LeFort fractures. They rarely occur as an isolated fracture. Some have advocated dividing these fractures into specific groups based on the location of the fracture in relationship to the maxillary alveolus, teeth, and palatal midline (Fig. 10).

Diagnosis

A palatal fracture should be expected when there is disruption of the palatal and gingival mucosa. Other clinical signs may include a change in occlusion, mobility of alveolar segments, or a palpable bony step in the palatal vault (Fig. 11). A CT scan with axial and coronal slices should be acquired to confirm the diagnosis.

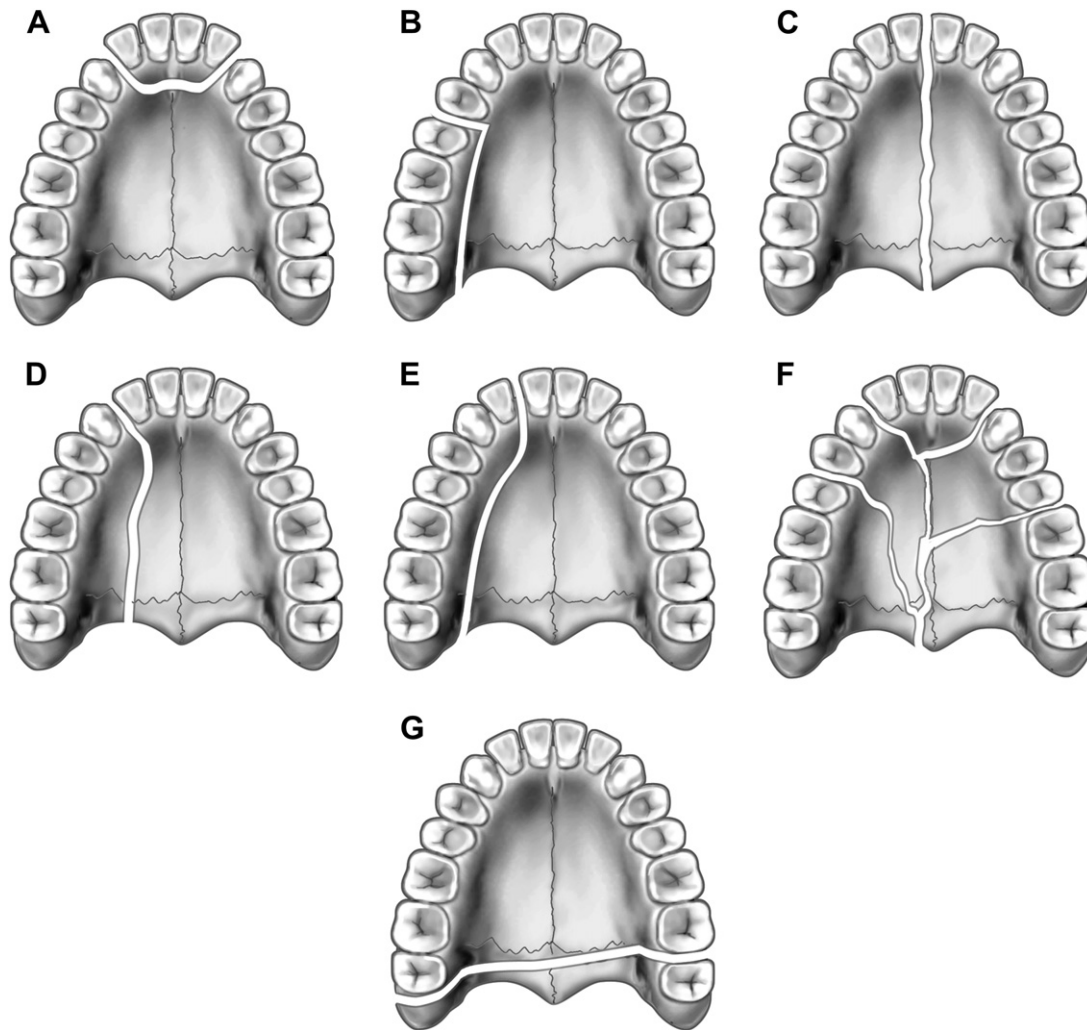


Fig. 10 Types of palatal fractures: (A) type Ia, anterior alveolus; (B) type Ib, posterolateral alveolus; (C) type II, sagittal; (D) type III, parasagittal; (E) type IV, para-alveolar; (F) type V, complex/comminuted; and (G) type VI, transverse. (From Hendrickson M, Clark N, Manson P, et al. Palatal fractures: classification, patterns and treatment with rigid internal fixation. *Plast Reconstr Surg* 1998;101(2):319; with permission.)

Surgical treatment and postoperative care

Treatment of palatal fractures may involve a combination of arch bars, rigid internal fixation and palatal or occlusal splints. Consideration of associated mandibular and midfacial fractures should determine the approach and sequencing. The goal of treatment is to reproduce the preinjury occlusion. Hendrickson and colleagues (1998) described a step-wise treatment algorithm based on the fracture type. Those fractures involving the

anterior alveolus (type Ia) and posterolateral alveolus (type Ib) are treated with a segmental arch bar spanning the teeth adjacent to the fracture lines. In addition, when possible, miniplates can be placed in the region of the nasomaxillary buttress (Ia) and zygomaticomaxillary buttress (Ib) to further stabilize these fractures. A period of 2 to 4 weeks of MMF is recommended. Types II, III, IV, and VI fractures are treated with a combination of arch bars and rigid internal fixation. An arch bar is first loosely applied to achieve preliminary alignment of the segments. Next, the patient is placed into MMF to determine occlusal accuracy. The patient is then released from MMF and the palate is exposed via an existing laceration or longitudinal incision, paying close attention to the protection and preservation of the greater palatine vessels. After exposing the fracture, a minimum of 2 plates are applied to prevent posterior splaying of the segments. This procedure is best performed while applying medial pressure from the lateral sides of the segments. The wound is then closed and the patient is placed back into MMF. The nasomaxillary and zygomaticomaxillary buttresses are then exposed, with rigid internal fixation as necessary. The occlusion is checked and the patient is placed into MMF for 2 to 4 weeks. Comminuted (type V) fractures are treated with a palatal acrylic splint. Incisions should be avoided to preserve the blood supply to the bony



Fig. 11 Type II palatal fracture showing laceration of palatal mucosa and asymmetry about the midline with bony displacement.

fragments. The splint is fabricated after preoperative model surgery on dental casts to determine the desired occlusal relationship and then wired into place for approximately 6 weeks. These splints can be placed with or without arch bars. Alternatively, for all fracture types, practitioners familiar with model surgery can fabricate an occlusal splint before surgery. Here, fracture reduction relies on the clinician's preoperative estimate of the preinjury occlusion. The splint should be left in for a minimum of 6 weeks and should be supported by rigid internal fixation at the LeFort I to III levels, as necessary.

Postoperatively, patients should be kept on a soft diet for 4 to 6 weeks. When arch bars are in place, guiding elastics can be used after MMF is released.

Zygomaticomaxillary complex fractures

Anatomy

The zygomas are a major determinant of facial symmetry and form. The zygoma has been described as a quadrangular bone with 4 processes. It articulates with the maxilla, temporal, sphenoid, and frontal bones (Fig. 12). The sutures created by these articulations are common points of fracture (Fig. 13).

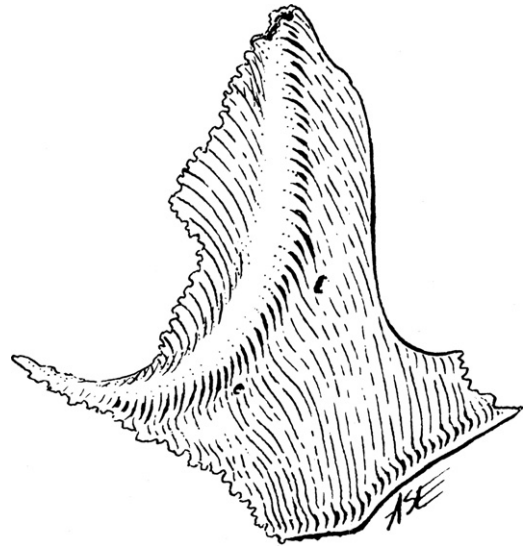


Fig. 12 The articulations of the zygoma: frontal, temporal, orbital, and maxillary. (From Ellis E. Fractures of the zygomatic complex and arch. In: Fonseca RJ, editor. Oral and maxillofacial trauma. vol. 2. St Louis (MO): WB Saunders; 2005. p. 571; with permission.)

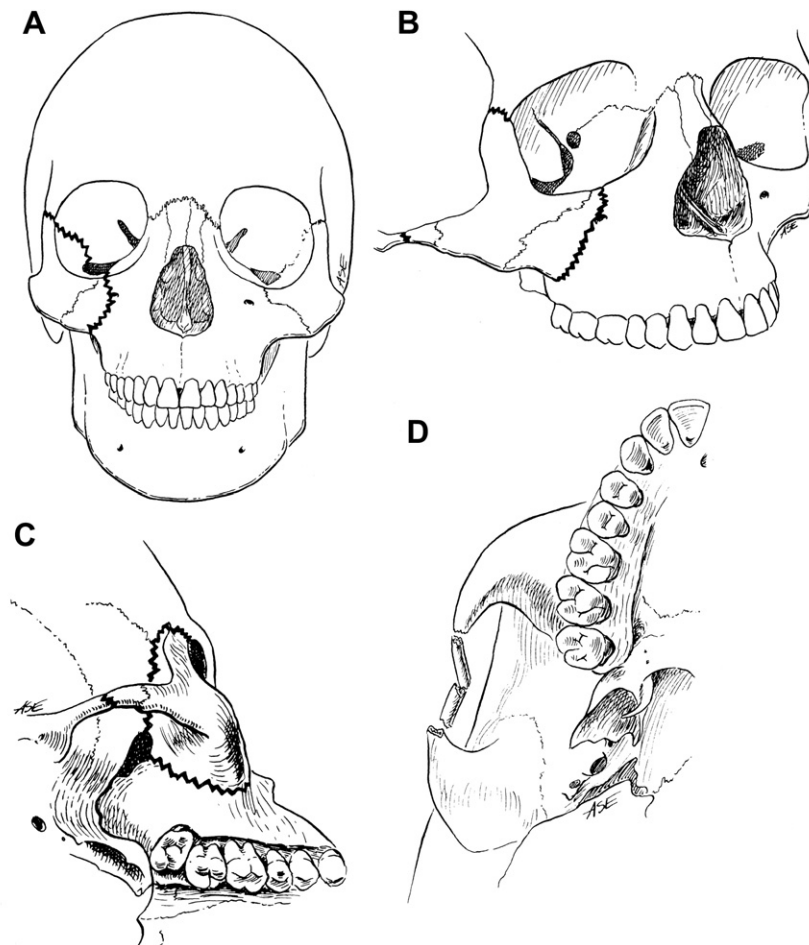


Fig. 13 The common points of fracture for the zygomaticomaxillary complex. (A) Frontal view of skull showing fracture medial to zygomaticomaxillary suture and along zygomaticosphenoid suture. (B) Oblique view showing fractures through the zygomaticofrontal suture and posterior to the zygomaticotemporal suture. (C) Temporal view showing fractures extending from the inferior orbital fissure both superiorly and inferiorly through the zygomatic buttress of the maxilla. (D) Inferior view showing fracture through the zygomatic arch. (From Ellis E. Fractures of the zygomatic complex and arch. In: Fonseca RJ, editor. Oral and maxillofacial trauma. vol. 2. St Louis (MO): WB Saunders; 2005. p. 573; with permission.)

The anatomy of the zygoma and its 4 articulations described earlier have led to terminology such as zygomatic complex and zygomaticomaxillary complex (ZMC) to describe fractures of this region. Appropriate reduction of the zygomaticosphenoid, zygomaticofrontal, and zygomaticomaxillary articulations are critical to the correct alignment of the fractured zygomatic complex. Because of the multiple articulations of the zygoma and proximity to the orbit, it has been estimated that 76% of fractures involving the zygoma also involve a portion of the orbital wall or floor.

In addition to providing the lateral prominence to the midface, the zygoma serves as a point of origin of the masseter muscle, as well as a point of attachment for the zygomaticus and temporalis muscles. When the zygomatic arch is severely comminuted or the fractured segments impinge on the temporal muscle, spasm can occur and lead to trismus. Fractures of the zygomatic arch can also limit mouth opening by impeding the movement of the coronoid process of the mandible.

Classification

Various classification schemes have been devised over the years to address the type and treatment of ZMC fractures. Classically, Knight and North described a scheme based on the

rotation and displacement of ZMC fractures. Manson described a scheme derived from a CT scan of the patient and classified the fracture pattern as a low-energy, medium-energy, or high-energy fracture. Zingg reviewed 1025 cases and reported a classification system that separated the type of fracture based on the site and degree of fragmentation.

We prefer the classification scheme by Zingg, which separates the fractures into 3 categories: types A, B, and C (Fig. 14). Type A fractures are broadly classified as incomplete zygomatic fractures and further subdivided into 3 categories: the isolated zygomatic arch fracture (A1), lateral orbital wall fracture (A2), and an infraorbital rim fracture (A3). Type B fractures are defined as a monofragment zygomatic fracture, in which the 4 articulations of the malar bone are fractured and may be displaced. Type C fractures encompass the same fracture pattern as the type B fractures, with the additional finding of fragmentation of the malar bone processes and malar body.

Diagnosis

The decision to operate on fractures of the ZMC cannot solely be based on where the fracture falls within a certain classification scheme. Limitations in mandibular movement, abnormalities in facial symmetry/contour, disturbance of the visual

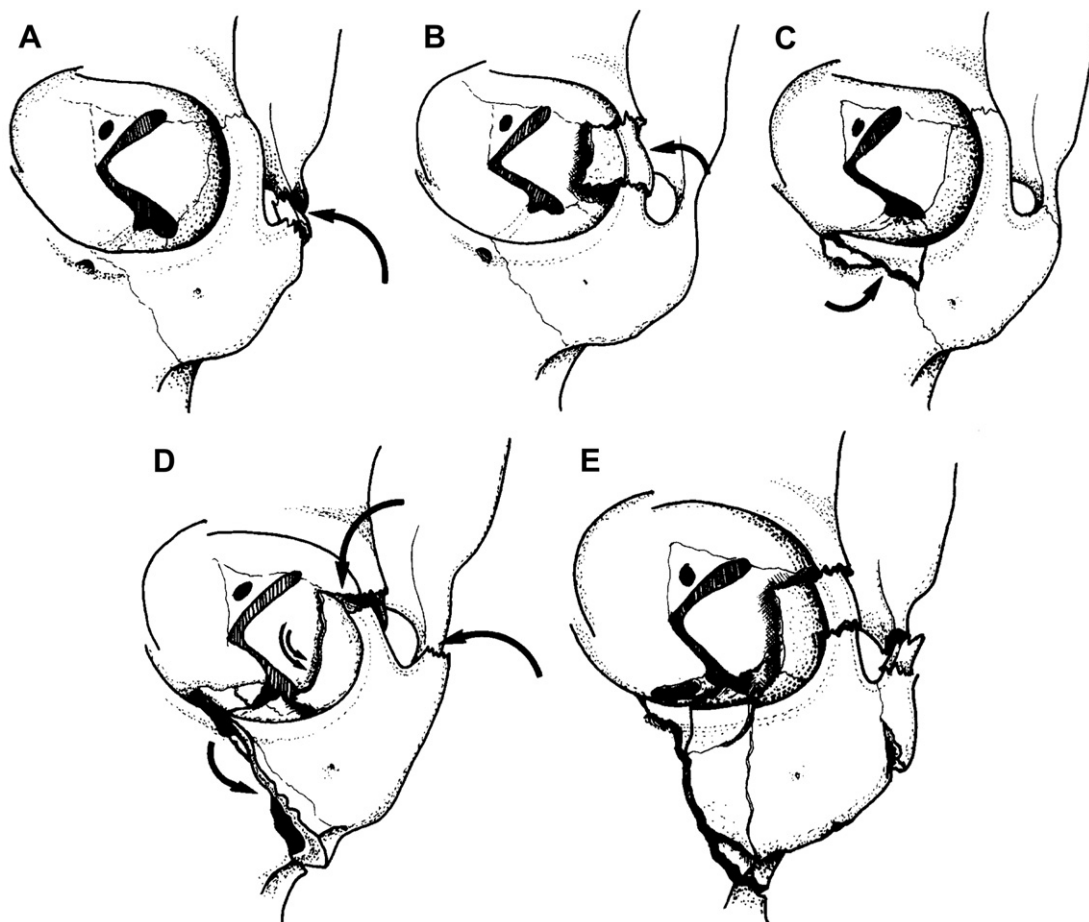


Fig. 14 Zygomaticomaxillary complex fracture types: (A) type A1, isolated zygomatic arch fracture; (B) type A2, isolated lateral orbital wall fracture; (C) type A3, isolated infraorbital rim fracture; (D) type B, tetrapod fracture; (E) type C, multifragment zygoma lateral orbit complex fracture. (From Zingg M, Laedrach K, Chen J, et al. Classification and treatment of zygomatic fractures: a review of 1025 cases. *J Oral Maxillofac Surg* 1992;50:778; with permission.)

fields, globe displacement or disruption, and involvement of the orbital floor should be considered when deciding on an operative treatment plan.

With the advancement and availability of three-dimensional CT imaging much of the difficult determination of the fracture pattern and level of displacement has been solved (see Fig. 18B). In areas in which CT imaging may not be readily available, plain films can still provide a wealth of information. When combined with an appropriate clinical examination, the Waters, Caldwell, and submentovertex views can provide sufficient information to confirm a clinical diagnosis. The Water view is generally best for evaluation of inferior orbital rim, the maxillary portion of zygoma, and the maxillary sinus. The Caldwell view is useful for diagnosis of fractures of the frontal process of the zygoma and zygomaticofrontal suture. The submentovertex is useful for evaluation of the zygomatic arch and its articulations. Patient positioning is critical, and having an experienced team obtain the radiographs is an important consideration.

Surgical treatment

The primary goals of providing surgical treatment are to restore the preinjury form and function of the patient. Individual assessment of the fractures is critical to the determining the appropriate approach and fixation technique. Repairing fractures of the midface should be attempted in such a manner as to decrease the morbidity of the sustained trauma.

Minimalist approaches for reduction of the zygomatic complex may avoid the need for further surgical treatment if the bones are stable after reduction. The Gillies temporal approach and Keen maxillary vestibular approach offer simple ways to access the zygoma and zygomatic arch. The Gillies approach uses a temporal incision approximately 2.5 cm superior and anterior to the helix of the ear. The incision should fall posterior to the hairline to avoid a noticeable scar. The incision is carried through skin and subcutaneous tissue down to the temporal fascia. The temporal fascia is then incised, exposing the temporalis muscle beneath. An elevator such as a number 9 periosteal elevator or freer elevator is then placed between the muscle and fascia and swept back and forth to approach the medial side of the zygoma and zygomatic arch. A more rigid instrument such as a urethral sound or Rowe zygomatic elevator may then be inserted to apply force and reduce the bony segments. The Keen approach is an intraoral version of this reduction technique. Here, an incision is made

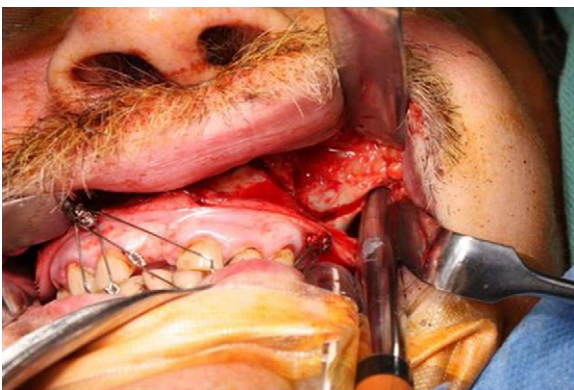


Fig. 15 Keen approach for ZMC fracture reduction.



Fig. 16 Set-up for Carroll-Girard screw. A trochar and drill are used to make a hole in the body of the zygoma. The Carroll-Girard screw is then inserted.

over the zygomatic buttress of the maxilla. A periosteal elevator is then inserted in a suprapariosteal plane and swept back and forth to approach the infratemporal surface of the zygoma. A larger, more rigid instrument is then inserted to reduce the bony segments (Fig. 15). The most direct approach for reduction of a depressed ZMC fracture is the percutaneous approach. Here, a small nick incision is made directly over the body of the zygoma. A bone hook can then be inserted behind the zygoma to apply reductive forces. Alternatively, a bone screw, such as a Carroll-Girard screw, can be inserted into the zygoma to aid with reduction (Fig. 16). This instrument is useful in that it allows for control of the zygoma in all directions of space (Fig. 17). The disadvantage of using percutaneous methods is the potential for scar formation in an esthetic area.

More invasive approaches may be indicated to address unstable fractures that are difficult to reduce. In these cases, rigid internal fixation with 1.5-mm to 2.0-mm plates is indicated (Fig. 18). We recommend exposure of at least 3 of the 4 zygoma articulations with fixation as necessary to ensure proper alignment and prevent unwanted rotation of the zygomatic complex. Proper reduction of the zygomaticosphenoid component offers the most accurate intraoperative indicator of proper positioning. Lacerations that have been created during the traumatic event may be appropriate access points if they can be used safely without additional damage to adjacent vital structures. The following is a brief discussion of the separate techniques to approach fractures of the ZMC.



Fig. 17 Direct percutaneous reduction method using Carroll-Girard screw.



Fig. 18 Surgical treatment of right ZMC fracture in patient sustaining gunshot injury to right temporal region. (A) Initial presentation of patient with gunshot injury to left temporal region that exited right temporal region. Patient had significant intracranial injury, and intraventricular drain was placed because of increased intracranial pressure. (B) 3D CT reconstruction of type C ZMC fracture with inferolateral displacement of the zygoma and comminution of the zygomatic arch. (C) Appearance 2 weeks after injury. Noticeable prominence of right lateral orbit and zygoma because of inferolateral displacement of ZMC. Patient underwent frontal and temporal craniectomy and a tracheostomy and had been medically stabilized. (D) Surgical exposure of zygomaticofrontal region via upper blepharoplasty approach. (E) Reduction and fixation of ZF with 1.5-mm plate. (F) Surgical exposure of infraorbital rim through lower eyelid crease approach. (G) Reduction and fixation of infraorbital rim fractures with 1.5-mm plates. (H) Exposure of right zygomaticomaxillary buttress via maxillary vestibular incision. (I) Reduction and fixation of ZM with 2.0-mm L-plate. (J) Appearance of patient 1 week after reconstruction of ZMC fracture. Note the improved projection of the right zygoma and lateral orbit. A frost stitch was placed in the lower lid to help prevent ectropion. (K) Postoperative 3D CT showing reduction of right ZMC. The comminuted zygomatic arch fracture was not addressed after consultation with neurosurgery because of intracranial injuries.

Coronal approach

A coronal flap is a useful approach when extensive exposure of the zygomatic arch, zygoma, and orbit is indicated (Fig. 19). The incision should be designed in such a way that the scar is hidden within the hairline or presumed future hairline. When wide access is indicated and approach to the infraorbital rim and lower portions of the zygoma, the incisions can be extended in a preauricular fashion to allow for further dissection and reflection of the flap.

Supraorbital eyebrow approach

A 2-cm incision placed over the lateral portion of the eyebrow allows rapid access to the lateral orbital rim and

the zygomaticofacial suture. This is a quick and rapid approach, and if eyebrows are present, the scar is usually well hidden. The incision is small, and if greater access is required to the ZF area, then additional approaches may be indicated.

Upper blepharoplasty approach

Using a natural skin crease in the lateral upper eyelid allows the surgeon to gain access to the ZF suture with minimal postoperative potential for unaesthetic scar formation (see Fig. 18D, E). In addition to the potential benefits to esthetics, this approach allows for greater access to the lateral orbital wall and the ZF region.

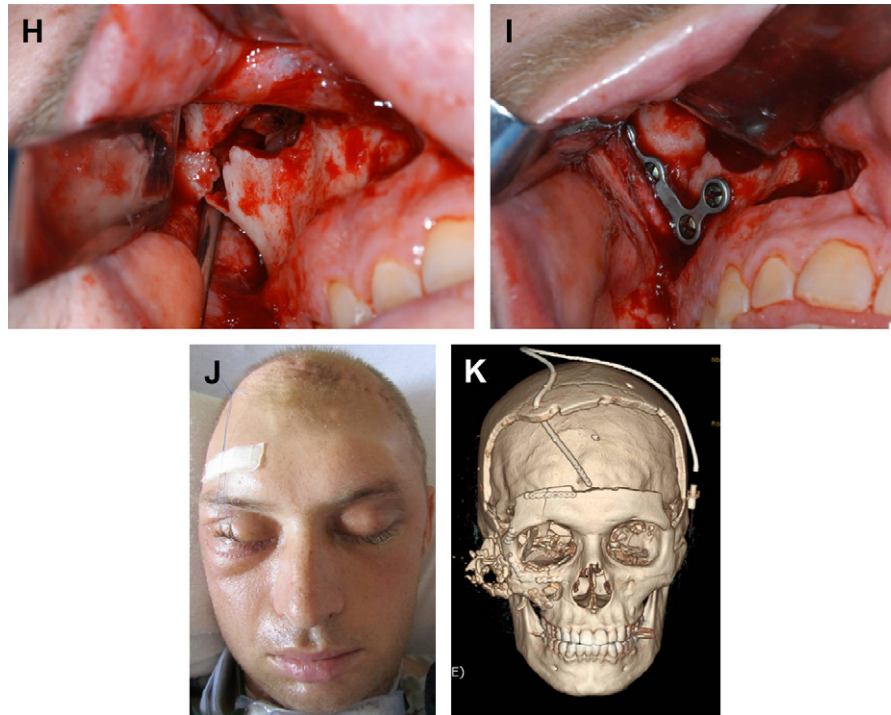


Fig. 18 (Continued)

Maxillary vestibular approach

Rapid and esthetic access to the anterior portions of the midface and zygomaticomaxillary buttress can be obtained through this approach (see Fig. 18H, I). Many surgeons refer to this approach as a free incision, because of the intraoral scar that is created. The ability to visualize the inferior orbital rim, zygoma, and anterior maxilla allows for access without significant difficulty in those areas.

Lower eyelid approaches

When access is needed to explore and restore fractures of the orbital walls and floor, 3 surgical options are available, depending on the access required and familiarity with the technique. The subciliary, lower eyelid crease (see Fig. 18F, G), and transconjunctival approaches are the most commonly used methods. The decision to approach the internal orbital fractures and their reconstruction options are discussed later. It is prudent for the surgeon to observe the patient for 24 hours if there is any concern about postoperative ophthalmologic complications.

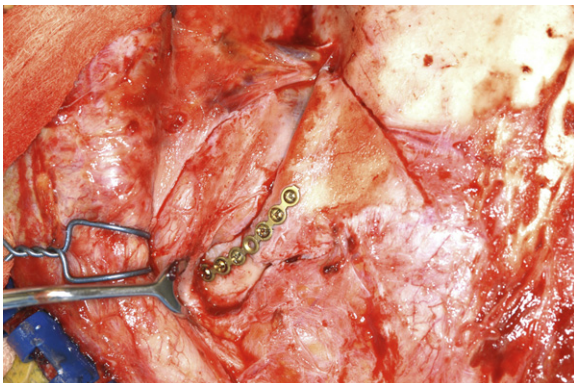


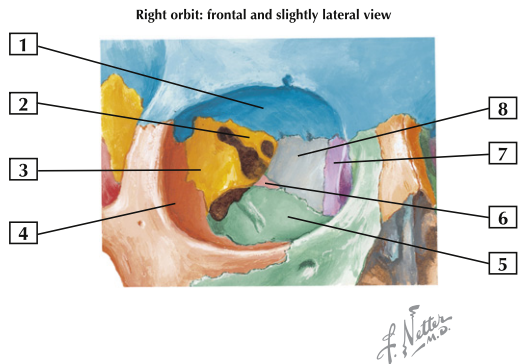
Fig. 19 Reduction and fixation of right zygomatic arch via coronal approach.

Orbital fractures

Anatomy

Seven bones make up the bony orbit: the maxillary, zygomatic, frontal, ethmoidal, lacrimal, palatine, and sphenoidal (Fig. 20). Its volume averages 30 mL. The orbital rims are made of dense cortical bone that thin considerably, extending posteriorly. Behind the superolateral rim is the lacrimal fossa. The nasolacrimal sac is about 4 mm posterior to the inferior rim and is housed behind the lacrimal crest. The inferior half of the medial wall, known as the lamina papyracea, is from 0.2 to 0.4 mm thick. It separates the orbit from the ethmoid air cells. The anterior and posterior ethmoidal foramina are located in the superior half of the medial wall approximately 24 mm and 36 mm posterior to the anterior lacrimal crest. The orbital floor is bordered laterally by the inferior orbital fissure, which gives rise to the infraorbital groove. This groove extends anteriorly and exits approximately 5 mm inferior to the infraorbital rim at its foramen. The floor of the orbit is also thin and is contiguous with the roof of the maxillary sinus. It is the most commonly fractured region in orbital trauma. The roof of the orbit separates the frontal lobe and sometimes the frontal sinus from the orbit. This wall along with the lateral wall is thicker and less likely to fracture. In the superoposterior aspect of the orbit is the superior orbital fissure and the opening of the optic canal. It is important to know the important structures associated with the fissures and foramina of the orbit (Table 1). An understanding of safe dissection distances when reconstructing the orbit prevents unwanted injury to vital structures (Table 2).

At the lateral aspect of the orbit is the Whitnall tubercle. The Whitnall tubercle lies 1 cm below the frontozygomatic suture and 3 to 5 mm posterior to the lateral orbital rim. The lateral horn of the levator aponeuroses, the lateral canthal tendon, Lockwood ligament (inferior suspensory ligament), and fine check ligaments of the lateral rectus muscle insert on this



1. Frontal bone
2. Lesser wing of the sphenoid
3. Greater wing of the sphenoid
4. Zygomatic bone
5. Maxillary bone
6. Palatine bone
7. Lacrimal bone
8. Ethmoid bone

Bones creating the orbital margin include:

- Frontal
- Zygomatic
- Maxilla

Walls of the Orbit

Superior	Frontal (orbital plate) Lesser wing of sphenoid
Inferior	Maxilla Zygomatic Palatine (orbital process)
Medial	Ethmoid (lamina papyracea) Lacrimal Sphenoid Maxilla
Lateral	Zygomatic Greater wing of sphenoid

Fig. 20 Bony anatomy of the orbit. (Netter illustration from www.netterimages.com. © Elsevier Inc. All rights reserved.)

tubercle. The medial canthal tendon (MCT) is formed by the pretarsal portions of the orbicularis oculi, where the upper and lower lids meet. It is divided into a superficial and deep portion by the lacrimal sac. The deeper portion is known as the Horner

muscle (pars lacrimalis) and attaches to the posterior lacrimal crest. The superficial portion has 2 legs and inserts onto the frontal process of the maxilla.

Classification

Many classification schemes have been proposed for orbital fractures. We find it most useful to describe orbital fractures by their location and general category: linear, blow-out, or blow-in.

Linear

The periosteum remains intact in a linear fracture. There is no herniation of orbital contents and usually no defect is appreciated. There may be a slight increase in orbital volume with delayed enophthalmos.

Table 1 Orbital fissures/canals and their contents	
Location	Contents
Superior orbital fissure: lesser and greater wings of sphenoid	Motor nerves: III (superior and inferior divisions), IV (trochlear), VI (abducens) Sensory nerves: V ₁ (frontal, lacrimal, nasociliary), sympathetic fibers Vessels: superior ophthalmic vein, anastomosis of recurrent lacrimal and middle meningeal arteries
Inferior orbital fissure: greater wing of sphenoid; palatine, zygomatic, and maxillary bones	Sensory nerves: V ₂ (infraorbital and zygomatic), parasympathetic branches of pterygopalatine ganglion Vessel: inferior ophthalmic vein and branches to pterygoid plexus
Optic canal: lesser wing of sphenoid	Optic nerve, meninges, ophthalmic artery, sympathetic fibers
Anterior ethmoid canal: frontal and ethmoid bones	Nerve: anterior ethmoid becomes dorsal nasal Vessel: anterior ethmoid artery
Posterior ethmoid canal: frontal and ethmoid bones	Nerve: posterior ethmoid Vessel: posterior ethmoid artery
Nasolacrimal fossa: lacrimal and maxillary bones	Nasolacrimal sac and duct

From Ochs MW. Orbital and ocular trauma. In: Miloro M, editor. Peterson's principles of oral and maxillofacial surgery. 2nd edition. Hamilton (Canada): 2004. p. 464; with permission.

Table 2 Distance of vital orbital structures from bony landmarks		
Structure	Reference Landmark	Mean Distance (mm)
Midpoint of inferior orbital fissure	Infraorbital foramen	24
Anterior ethmoidal foramen	Anterior lacrimal crest	24
Superior orbital fissure	Zygomaticofrontal suture	35
Superior orbital fissure	Supraorbital notch	40
Optic canal (medial aspect)	Anterior lacrimal crest	42
Optic canal (superior aspect)	Supraorbital notch	45

From Ochs MW. Orbital and ocular trauma. In: Miloro M, editor. Peterson's principles of oral and maxillofacial surgery. 2nd edition. Hamilton (Canada): 2004. p. 465; with permission.

Blow-out

This fracture is the most common and is described as a fracture limited to 1 wall up to 2 cm in diameter. This fracture usually occurs in the orbital floor, with displacement of orbital contents into the underlying maxillary sinus. These fractures have been described with medial and lateral walls as well. A complex blow-out fracture involves 2 or more walls, with medial wall fractures accompanying floor fractures more commonly. The complex classification is greater than a 2-cm defect and can extend to the posterior orbit and involve the orbital canal.

Blow-in

Blow-in fractures involve displacement of a wall or portion of a wall into the orbit. These fractures can occur in any wall but they occur most commonly in the roof. A dural tear should be suspected with a blow-in fracture of the orbital roof.

Diagnosis

CT imaging is an excellent tool to evaluate the bony walls of the orbits. Sagittal views are beneficial when looking at the posterior extent of fractures in the floor and roof of the orbits, where coronal views may be the most beneficial in evaluating the floor and medial walls (Fig. 21).

Periorbital lacerations should be closely examined for tarsal plate involvement and if occurring in the medial lids, injury to the lacrimal apparatus should be suspected. This condition can be further evaluated at the bedside or, when necessary, in the operating room. Fat herniation from the superior lid may indicate levator disruption, and widening of the canthal angles suggests canthal tendon disruption. Palpation of the orbits should be performed, but periorbital edema makes it difficult to appreciate step-offs. Paresthesia of the infraorbital distribution may indicate orbital floor involvement.

Clinical evaluation of patients with orbital trauma should always include an ophthalmologic examination. Assessment of visual acuity, extraocular movements and pupillary responses should be completed. In addition, a fundoscopic examination may help identify injuries to the globe. An accurate assessment of enophthalmos may be masked by edema until resolution of swelling. Enophthalmos may be best appreciated from a bird's

eye view looking down from the head of the bed (Fig. 22). Diplopia, when present, should be categorized as either monocular or binocular. Monocular diplopia may be caused by lens dislocation or opacification. Binocular diplopia can result from restricted mobility, edema, muscle injury, or neural injury. The conscious patient should be asked to go through all extraocular movements before and after any surgical intervention (Fig. 23). Limitation of movement in any direction should be noted. A forced duction test can also be performed when restriction in eye movement exists. Here, tissue forceps are used to grasp the inferior rectus and moved superiorly to assess for muscular entrapment (Fig. 24). A pocket Snellen chart may be used to assess the visual acuity of the patient. Remember to have the patient wear their corrective lenses for this examination. Pupillary reactivity to light, size, and shape should be noted to rule out anisocoria and irregularly shaped pupils.

Ophthalmologic consultation should always be considered with orbital trauma, but certain signs may suggest urgent consultation. Lacerations of the palpebral conjunctiva and tear drop-shaped pupils may suggest globe perforation (the apex of the pupil points to the side of perforation). These findings warrant elevation of the bed head, analgesics, antiemetics, and avoiding Valsalva maneuvers with emergent consultation. Signs of hyphema, traumatic mydriasis, and traumatic iridodialysis should also warrant immediate consultation. A retrobulbar hematoma should be suspected in patients showing extreme pain, visual impairment, and proptosis after ocular trauma. This condition warrants immediate surgical intervention via a lateral canthotomy with inferior cantholysis. Delay may result in permanent blindness.

Surgical treatment and postoperative care

Before surgical intervention is initiated, contraindications to orbital fracture repair should be reviewed. Hyphema, globe perforation, and blindness warrant consultation and recommendations from ophthalmology before surgery. Treatment may also need to be delayed based on the patient's systemic condition. Barring these conditions, correction of orbital injuries can be classified as functional or cosmetic. Diplopia, decreased visual acuity, muscular entrapment, and hematoma are all functional reasons for surgical correction. Cosmetic

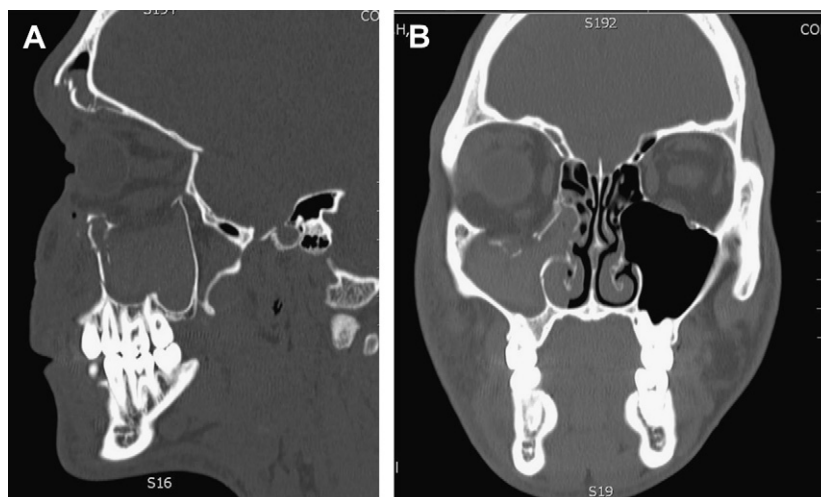


Fig. 21 CT imaging in the evaluation of orbital fractures. (A) Sagittal view showing posterior extent of orbital floor fracture. (B) Coronal view showing inferior displacement of right orbital floor with increased orbital volume.



Fig. 22 Bird's eye view of patient with orbital floor fracture shown in Fig. 21, showing enophthalmos of right eye.

indications include enophthalmos greater than 2 to 3 mm or fracture exceeding 50% of the orbital floor with fat herniation into the antrum. In these cases, early intervention leads to greater success in preventing future enophthalmos or functional deficits once swelling has resolved.

Several approaches have been described for access to the inferior rim and orbital floor, including (Fig. 25):

- Transconjunctival
- Subciliary
- Lower lid
- Infraorbital
- Existing lacerations

The transconjunctival approach offers the advantage of the lack of visible scarring and reduced incidence of ectropion. However, it does have a higher incidence of entropion compared with the other approaches. When combined with the

release of the lower limb of the lateral canthal tendon, this approach provides excellent visualization for exploration of the orbital floor (Fig. 26A). Closure of this incision must include a lateral canthopexy to prevent widening of the lateral canthal angle, ectropion, and increased scleral show (see Fig. 26C). The medial orbital wall may be approached through a coronal flap or through an extension of the transconjunctival access through the caruncle. This approach provides adequate exposure to the inferior half of the medial orbital wall. If fractures need to be addressed in the superior half of the medial orbital wall, a coronal or lateral nasal incision should be used.

Fractures of the orbital rims are typically addressed before reconstruction of the orbital floors and walls. Linear fractures of the orbital rims are reduced and fixated with miniplates (see Fig. 18F, G). The orbital floor can be reconstructed with several materials, including titanium plates, autogenous grafts, allograft, or alloplastic material (see Fig. 26). Examples of alloplastic products include silicone, Teflon, polyethylene, methyl methacrylate, and hydroxyapatite. Allogenic grafts include dura, cartilage, and bone. Autogenous grafting from the calvarium and anterior iliac crest are also popular. Specially designed titanium orbital floor plates are readily available in most midface trauma sets and may provide the simplest way to reestablish normal orbital volume.

Some advocate the use of a frost stitch through the lower lid after inferior orbital access to potentiate good inferior lid draping and help reduce wound contracture, which may cause ectropion (see Fig. 18J).

Postoperative CT imaging for orbital injuries should be used to document the final orbital reconstruction and confirm the appropriate placement of plates with avoidance of vital structures. Close follow-up of these patients after surgical repair is essential. The patient should be kept overnight after surgery to monitor for early postoperative complications. Postoperative examinations should rule out retrobulbar hematoma, superior orbital fissure syndrome, orbital apex syndrome, traumatic optic neuropathy, and corneal abrasions. A slit lamp examination can be used to evaluate for corneal abrasions.

Patients can be discharged from the hospital with ophthalmic bacitracin and oral analgesics. If injuries to the globe are

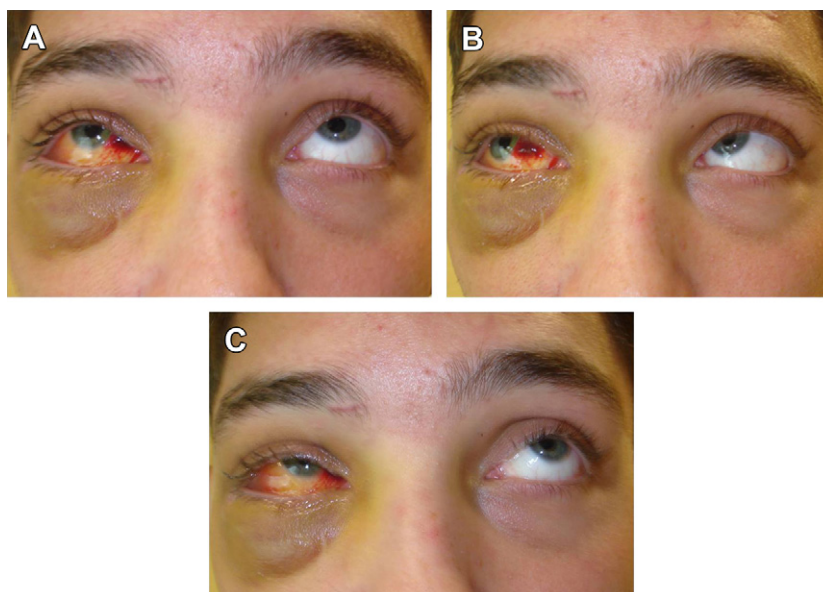


Fig. 23 (A–C) Patient having undergone orbital floor repair showing good range of motion in superior gaze.

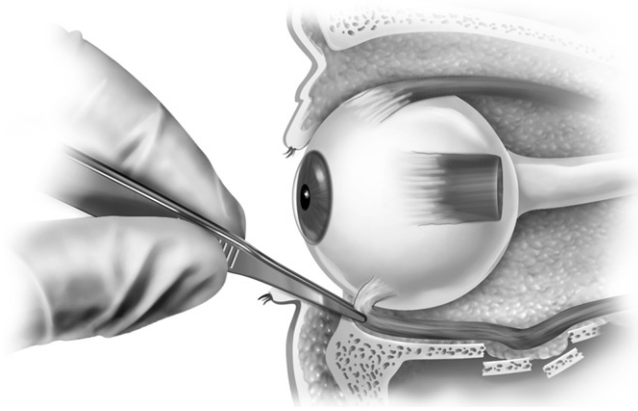


Fig. 24 A forced duction test can be performed to assess for muscular entrapment in patients with orbital floor trauma. (From Salin MB, Smith BM. Diagnosis and treatment of midface fractures. In: Fonseca RJ, editor. Oral and maxillofacial trauma. vol. 2. St Louis (MO): Elsevier; 2005. p. 677; with permission.)

present, postoperative management should be coordinated with an ophthalmologist. Weekly follow-up for the first 6 weeks should be established to monitor for postoperative lacrimal system injury. Monthly follow-up for 6 months should be undertaken to follow unresolved diplopia and monitor for the development of ectropion, entropion, or enophthalmos.

Nasal-orbital-ethmoid fractures

Anatomy

The interorbital space is bounded laterally by the medial walls of the orbits. The ethmoid air cells are within this space and average about 2.5 cm vertically and 1 cm in a transverse dimension. These air cells drain into the middle meatus, as does the nasofrontal duct from the frontal sinus. In the midline is the perpendicular plate of the ethmoid. The ethmoid, with the paired nasal bones, the frontal process of the maxilla, and

the nasal process of the frontal bone make up the medial walls. The MCT is a key anatomic structure in diagnosis of injury to this area. The average intercanthal distance is 28.6 mm to 33 mm for women and 28.9 to 34.5 mm for men (Fig. 27).

Classification

Markowitz and Manson have proposed a classification scheme for nasal-orbital-ethmoid (NOE) fractures (Fig. 28). This classification is based on the degree of bony comminution and the status of the MCT:

- Type I: MCT attachment is maintained to a large single nasoethmoidal fracture segment.
- Type II: MCT shows comminution, yet MCT maintains attachment to a sizable bony segment.
- Type III: MCT shows severe comminution of the bony segments and avulsion of the MCT.

Diagnosis

These fractures can be unilateral, bilateral, open, closed, simple, or comminuted. Axial and coronal CT imaging gives the extent of bony injury, and the clinical examination elucidates the soft tissue involvement, which may not be appreciated on imaging. Periorbital ecchymosis and edema are the most common clinical presentation (Fig. 29). Palpation of the orbital rims and the nasofrontal junction are required for obvious step-offs. Directional mobilization of the nasal complex and crepitus help determine the extent of the fracture. Widening and flattening of the nasal dorsum may be present but can be obscured by edema in the immediate posttraumatic period. Markowitz type I fractures typically do not show telecanthus, whereas type II and III injuries show some degree of telecanthus because of displacement of the MCT (Fig. 30). The intercanthal and interpupillary measurements can be compared with known norms. Widening of the canthal angles occurs with detachment of the MCT and is also an indicator of an NOE injury.

Several techniques can be used to evaluate the integrity of the NOE complex and the attachment of the MCT:

- In the bowstring test, tension is applied to the lateral canthal tendon while palpating the medial canthal region to assess for countertension or mobilization of a bony segment (Fig. 31).
- Bimanual palpation of the MCTs with an instrument inserted intranasally assesses for mobility of the medial canthus and its bony attachment.
- The Furness test uses forceps to grasp the skin overlying the MCT to determine bony attachment.

Intranasal examination should also be performed to determine the internal position of bony structures and to rule out the presence of CSF rhinorrhea.

Surgical treatment and postoperative care

Surgical access to the NOE region is most often accomplished through the coronal approach for its superior access and more desirable cosmetic results. Extension of lacerations in the area can also be considered as an approach to the fractures. Examples of other surgical approaches include the gullwing, open-sky,

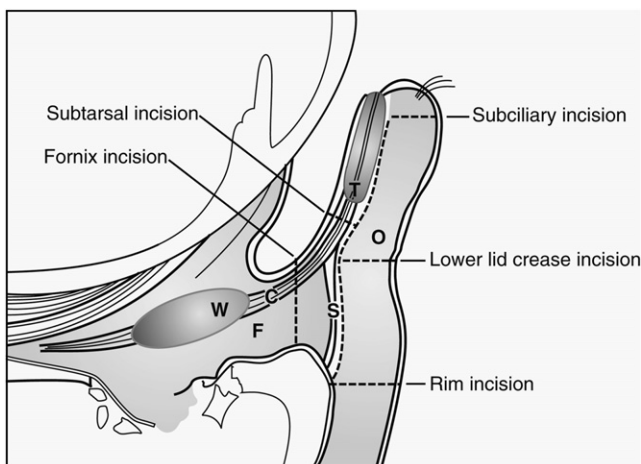


Fig. 25 Cross-sectional view of the inferior lid and various floor approach incisions. (From Ochs MW, Johns FR. Orbital trauma. In: Fonseca RJ, Marciani RD, Hendler BH, editors. Oral and maxillofacial surgery: trauma. vol. 3. Philadelphia: WB Saunders; 2000. p. 208; with permission.)

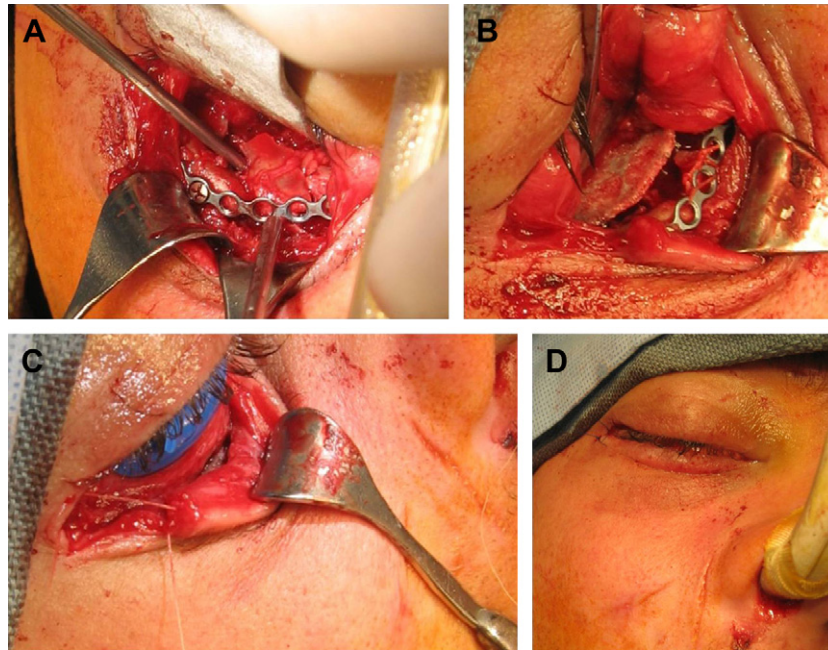


Fig. 26 Intraoperative photographs of orbital floor reconstruction for patient with orbital floor blow-out fracture shown in Figs. 21–23. (A) Fixation of orbital rim fractures with 1.3-mm plate through transconjunctival approach with lateral canthotomy and inferior cantholysis. (B) Reconstruction of orbital floor with Medpor Titan porous polyethylene. (C) Lateral canthopexy using 4-0 polydioxanone suture. (D) Appearance of lower eyelid and lateral canthal angle after closure of skin.

and butterfly approaches (Fig. 32). With these alternatives, significant extension is often necessary for access, and prominent scarring may occur. To achieve adequate bony fixation and repair of the medial canthal attachment, access to the medial orbital wall via a lower eyelid approach and maxillary vestibular incisions may be necessary.

Type I injuries can be treated both open and closed. Closed treatment involves manual reduction with use of an external nasal splint for 7 to 10 days. Open treatment involves accurate

reduction with miniplate fixation. If possible, attempts should be made to stabilize the bony segment along the nasofrontal and nasomaxillary junctions. Care should be taken to protect the medial canthal attachment. Markowitz type II and III fractures require transnasal wiring or suturing. After gaining surgical access, the medial canthal tendon is identified using tissue forceps. If the tendon is attached to a small fragment of bone (type II), a 30-gauge wire or 3-0 nylon suture may be used to secure this segment to adjacent stable bone. If this procedure is not possible, the wire or suture must be passed transnasally and secured to the contralateral side. It should be directed in a posterior and superior direction, to avoid widening of the nasal bones and blunting of the MCT. If the tendon is completely avulsed (type III), a wire or suture should be placed through the tendon with a mattress technique. It can then be attached to the medial crest on the opposite side or the other avulsed MCT (Fig. 33). Spinal needles or wire passing awls can be used through drilled holes to pass the wire or suture to the opposite side. Tightening the wire or cinching the suture over a titanium plate or screw is an effective way to secure its position. Slight overcorrection is indicated in this transnasal technique to avoid unaesthetic results. Orbital wall and floor fractures should be fixated before any canthopexy.

With severe comminution of the nasal bridge, a nasofrontal strut graft may be necessary to achieve adequate projection at the nasofrontal junction and nasal dorsum (see Fig. 33F–I). This graft may be autogenous, allogenic, or alloplastic and should be the last stage in NOE reconstruction. Suggested sequencing of these fractures is as follows:

- Surgical access
- Identification of the MCT and associated bone
- Reduction and reconstruction of the orbital rims
- Reconstruction of the medial orbital wall

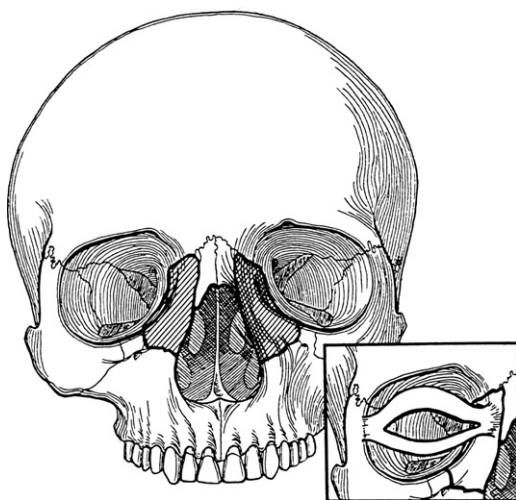


Fig. 27 The central nasoethmoid complex is highlighted; inset shows the bony attachment of the MCT. (From Salin MB, Smith BM. Diagnosis and treatment of midface fractures. In: Fonseca RJ, editor. Oral and maxillofacial trauma. vol. 2. St Louis (MO): Elsevier; 2005. p. 661; with permission.)

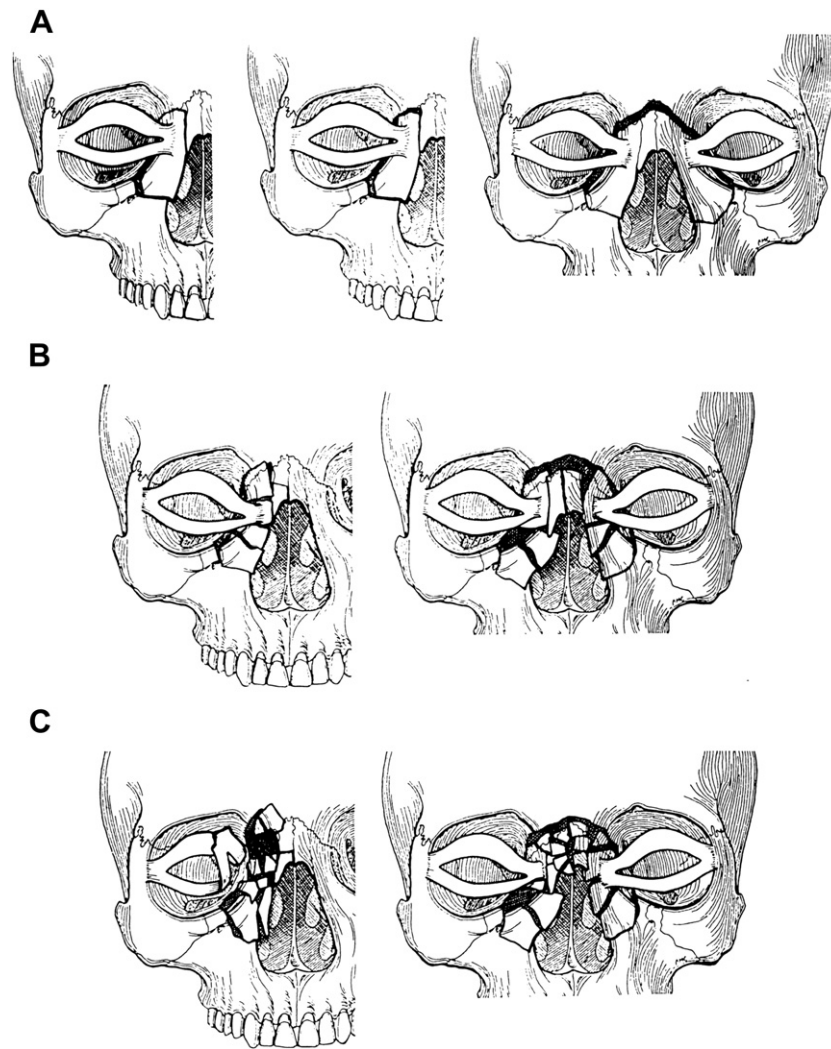


Fig. 28 Classification of NOE fractures: (A) type I, (B) type II, and (C) type III. (From Markowitz BL, Manson PN, Sargent L, et al. Management of the medial canthal tendon in nasoethmoid orbital fractures: the importance of the central fragment in classification and treatment. *Plast Reconstr Surg* 1991;87:843; with permission.)

- Transnasal canthopexy
- Reduction of septal fractures
- Reconstruction of nasal dorsum with external splinting as necessary
- Soft tissue adaptation

Postoperative CT imaging should be used to evaluate the reduction and fixation of NOE repair. After open reduction and internal fixation of NOE fractures, an external thermoplastic or Denver nasal splint may be used. Internal nasal splinting with an iodoform gauze packing is also helpful. If used, this packing should be removed in 7 days. We recommend the use of antibiotics and decongestants in the immediate postoperative period. Analgesics should be used as necessary.

Complications

Complications after midfacial trauma can occur with some frequency. These complications may include bleeding, malunion/nonunion, neurologic complications, ocular complications, lacrimal system complications, and CSF rhinorrhea/otorrhea.

Bleeding

The cause and initial management of bleeding in midface trauma were discussed earlier. Severe hemorrhage can occur intraoperatively and postoperatively. Intraoperative bleeding can be controlled under direct visualization with vessel ligation or electrocautery. If these measures are unsuccessful, gauze packing can be placed with a variety of local agents including local anesthetic with epinephrine and topical thrombin. Other products such as Floseal hemostatic matrix (Baxter Healthcare Corporation, Hayward, CA, USA), Avitene microfibrillar collagen (Davol Inc, Warwick, RI, USA), Gelfoam absorbable gelatin (Pharmacia and Upjohn Company, Kalamazoo, MI, USA), and Surgicel oxidized cellulose polymer (Ethicon, LLC, San Lorenzo, PR, USA) can be applied. If all of these attempts fail, selective embolization by an interventional radiologist should be performed. In the postoperative period, new-onset bleeding can be managed as in the initial management. When control is not possible, the patient should be taken to the operating room for control under direct visualization or to interventional radiology for selective embolization.



Fig. 29 Clinical presentation of patient 4 days after blast injury with panfacial trauma. Note the extensive NOE component with clinical features including orbital dystopia, telecanthus, periorbital edema, periorbital ecchymosis and lack of dorsal nasal projection.

Malunion/Nonunion

A malunion is defined as bone healing in an abnormal position. A nonunion is the cessation of healing without bony union. Delays in treatment because of coexisting disease, failure to diagnose, refusal by the patient of treatment, or lack of access to care can contribute to these complications. Failures in surgical treatment, such as inaccurate reduction and stabilization, can also be factors. Malunions and nonunions of mid-facial bones can cause both functional and cosmetic problems. If a malunion is present with no functional deficits (ie, malocclusion), surgical treatment should be based on the



Fig. 30 Patient with type III NOE fracture 2 weeks after injury, showing traumatic telecanthus and decreased dorsal nasal projection.

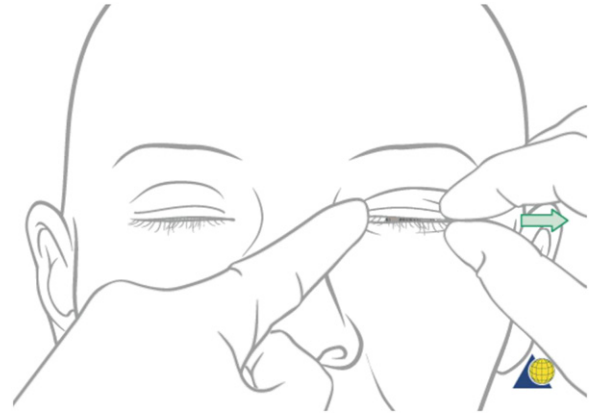


Fig. 31 Bowstring test: tension is applied laterally while palpating the MCT. Lack of resistance or movement of bony segments indicates an injury to the NOE complex. (From AO Foundation. Diagnosis of NOE fractures. Available at: <https://www2.aofoundation.org/wps/portal/surgery?showPageZdiagnosis&boneZCMF&segmentZMidface>; with permission. © AO Foundation/CMF Manual/2012.)

patient's desire to improve the cosmetic outcome. Surgical options include osteotomies with rigid internal fixation, alloplastic implants, and autogenous bone grafting. Soft tissue fillers can also be used for small-volume contour defects. Nonunions typically require more aggressive treatment. The fracture sites should be exposed, fibrous tissue debrided, and bones reduced and fixated in their proper position. Autogenous bone grafting may be necessary to bridge bony gaps and enhance healing. When serious functional deficits are noted, reoperation is indicated and the appropriate workup should be undertaken.

Neurologic complications

The infraorbital nerve is affected in greater than 50% of fractures involving the zygoma and orbit. The common signs of nerve injury include altered sensation/anesthesia of the upper lip, lower eyelid, lateral aspect of the nose, and the ipsilateral maxillary dentition and gingiva. Multiple studies have shown the relative recovery of infraorbital nerve function when appropriate reduction and fixation have occurred.

In addition to involvement of the infraorbital nerve, the frontal portion of the temporal branch and zygomatic branches of the facial nerve are most likely to be injured during the injury or its subsequent repair. Damage to the frontal branch can lead to permanent impairment of the frontalis muscle. Zygomatic and buccal branch involvement can lead to permanent paralysis of the eyelid. Iatrogenic injuries to this nerve can be prevented with careful surgical technique.

Ocular complications

Loss of vision in the postoperative orbit is a devastating complication. This loss of vision can be caused by retrobulbar hematoma, creating a compartment syndrome behind the globe that compresses the retinal artery, resulting in ischemia. A lateral canthotomy/cantholysis with drainage of the fluid collection is indicated immediately. Signs and symptoms include quickly declining visual acuity, pain out of proportion to presentation, proptosis, and an uncompressible globe. This

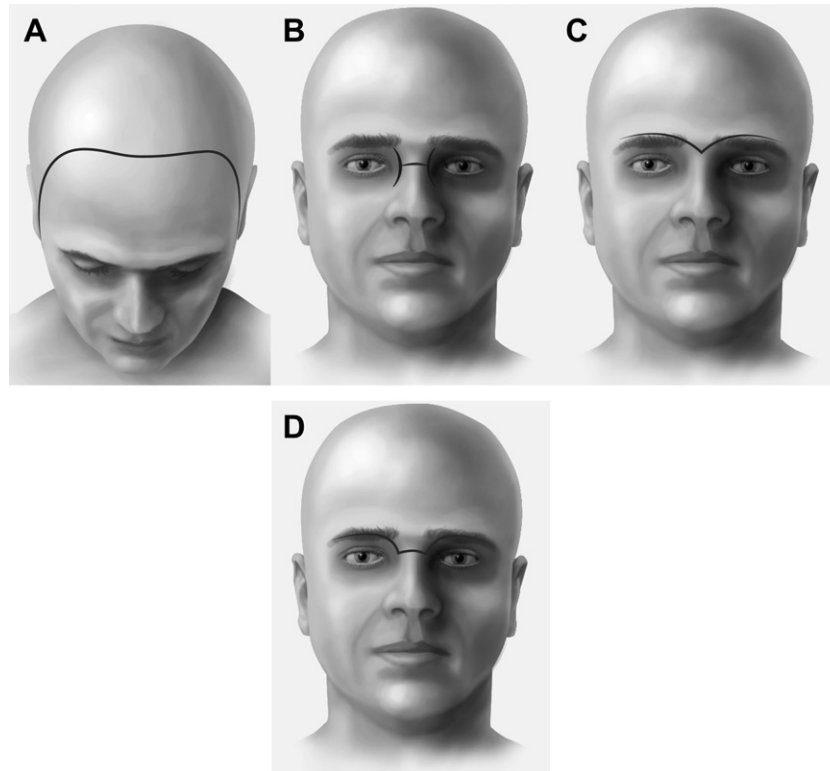


Fig. 32 Surgical access and incision to the nasofrontal region: (A) coronal, (B) open-sky, (C) gullwing, and (D) butterfly. (From Chuang SK, Dodson TB. Evaluation and management of frontal sinus injuries. In: Fonseca RJ, editor. Oral and maxillofacial trauma. vol. 2. St Louis (MO): Elsevier; 2005. p. 726; with permission.)

complication may also be encountered as a result of direct pressure on the optic nerve or its vasculature from surgical plates or graft placement too posteriorly in the floor. The inclination of the floor can drive this plate superiorly and encroach on these vital structures. The implanted/grafted material must be removed or readjusted immediately. For this reason, it is prudent to keep patients in the hospital for at least 24 hours after orbital reconstruction for routine visual checks. Close outpatient follow-up should also be instituted in the immediate postoperative period.

Diplopia is a common postoperative finding after orbital repair. Binocular diplopia is typically caused by residual post-traumatic or postoperative edema and disharmony of the extraocular movements. Entrapment of orbital soft tissue with a limitation in ocular motility, damage to the extraocular muscles, or an injury to cranial nerves III, IV, or VI may also be a cause. Therefore, good preoperative and postoperative evaluations are necessary for comparison. Immediately before surgical closure, a forced duction test should be performed to rule out anatomic restriction.

Enophthalmos occurs when increased orbital volume causes the globe to be displaced inferiorly and posteriorly. This displacement is typically only of cosmetic concern. However, if the vertical settling of the globe is greater than 1 cm, diplopia can result. Orbital floor fractures are the most common cause of enophthalmos in the patient with midface trauma. Correction involves reconstruction of the orbit as described earlier.

Epiphora is a postoperative complication that can arise as a result of ectropion, entropion, or injury to the nasolacrimal system. Scar contracture or poor reapproximation of the lateral canthal ligament may lead to ectropion. Scar contracture of the lower eyelid pulls the inferior punctum away from

the surface of the eye, compromising lacrimal drainage. This postoperative result may be avoided with proper selection of surgical access and good closure techniques. Transconjunctival incisions have the lowest incidence of postoperative ectropion and transcutaneous incisions have the highest. Modifications such as the midlower lid incision and the skin muscle flap version of the subciliary incision have lower incidence of ectropion. These modifications incorporate vertical stepping through different tissue layers and avoid full-thickness contracture. Imprecise reapproximation of the lateral canthal tendon may also cause ectropion. By overcorrecting its attachment to the periosteum in a vertical direction, this complication can be avoided. In entropion, the lower eyelid is rolled in against the globe. Obstruction of the inferior punctum leads to poor lacrimal drainage and epiphora.

Lacrimal system complications

A lacrimal drainage injury is a complication of both orbital and NOE injuries. Epiphora, as discussed earlier, is the most recognizable sign of this complication. A lacrimal drainage injury could be a result of failure to diagnose preoperatively, failure to reapproximate canaliculi that were injured with lower lid lacerations, or iatrogenic trauma inflicted during fixation and reconstruction. To evaluate the ducts, Jones I and Jones II tests can be performed. Jones I is performed by adding 2% fluorescein dye to the palpebral reservoirs and waiting 5 minutes for the dye to drain into the inferior meatus of the nose. If no dye is noted (negative), the patient should blow their nose or place their head in a forward position to facilitate drainage through the nose and not the nasopharynx. If there is still no sign of dye, the clinician

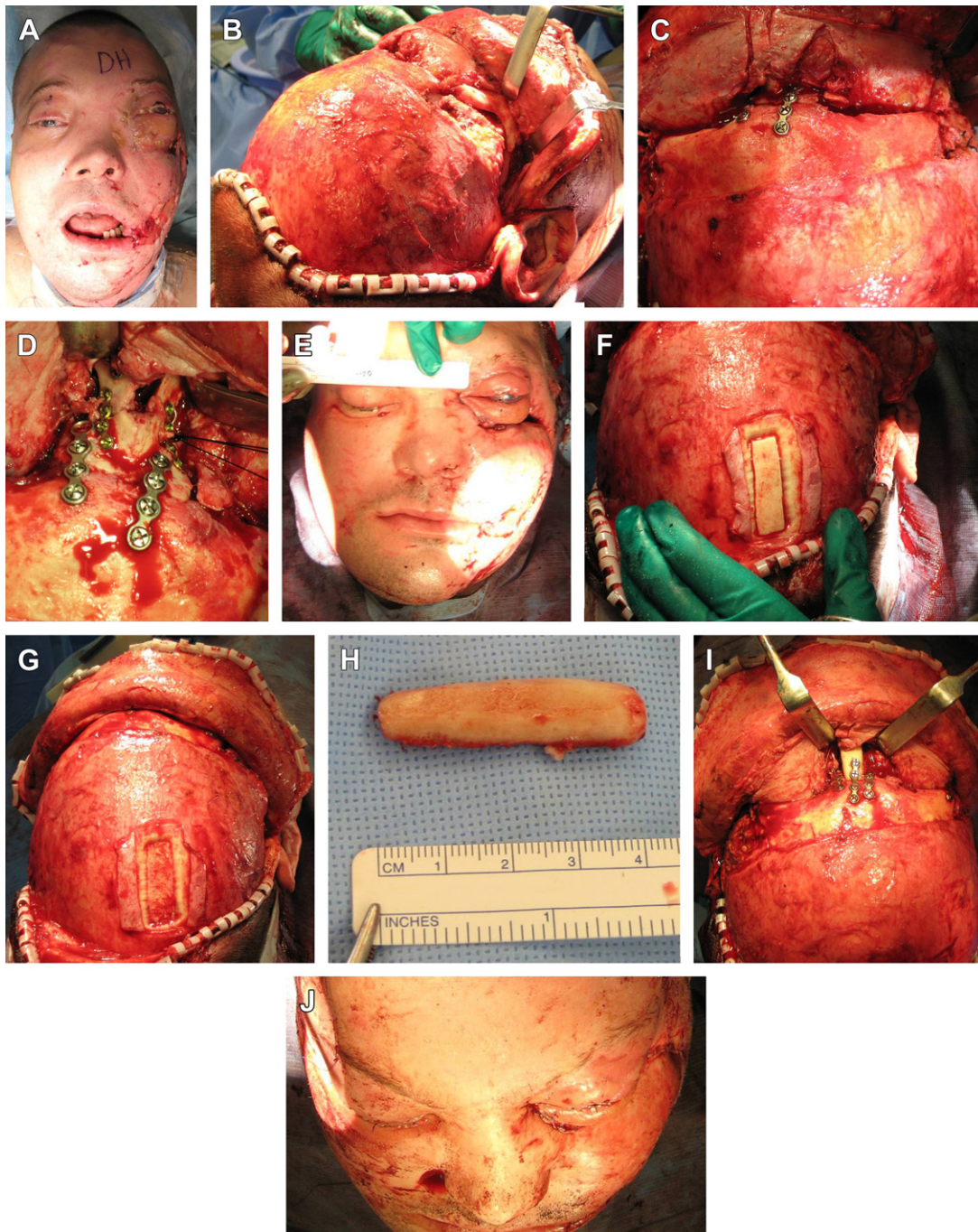


Fig. 33 Reconstruction of NOE complex in patient with comminuted type III fracture. (A) Appearance of patient in Fig. 29 14 days after injury. (B) Coronal flap with preauricular extension used to access nasofrontal region, lateral orbits, and zygomatic arches. (C) Reduction and fixation of fracture at nasofrontal suture with 2.0-mm miniplates. (D) Reduction and fixation of lateral nasal bones with 1.5-mm miniplates. 3-0 nylon suture placed through left MCT and passed transnasally and secured to plate on contralateral side. (E) Intraoperative assessment of intercanthal distance to confirm positioning. (F–H) Harvesting of split-thickness calvarium for dorsal nasal strut. (I) Autogenous nasal strut graft inset and fixated. (J) Appearance of NOE region before closure, showing decreased intercanthal distance and increased dorsal nasal projection.

should advance to the Jones II test. This test is used to ascertain the location of the obstruction. Here, the residual dye is first flushed from the lacrimal sac, and a cannula is inserted in the inferior canaliculus. The patient leans forward and saline is flushed into the system via the inferior canaliculus. If fluid appears in the nose with flushing, then a partial obstruction exists. If there is reflux noted at the

superior punctum, the obstruction is at or distal to the lacrimal sac.

Dacrocystorhinostomy (DCR) is a method for correcting injuries of the lacrimal system distal to the lacrimal sac. A bony window is created into the nose, and the lacrimal sac is connected to the nasal mucosa, thus bypassing the nasolacrimal duct (Fig. 34).

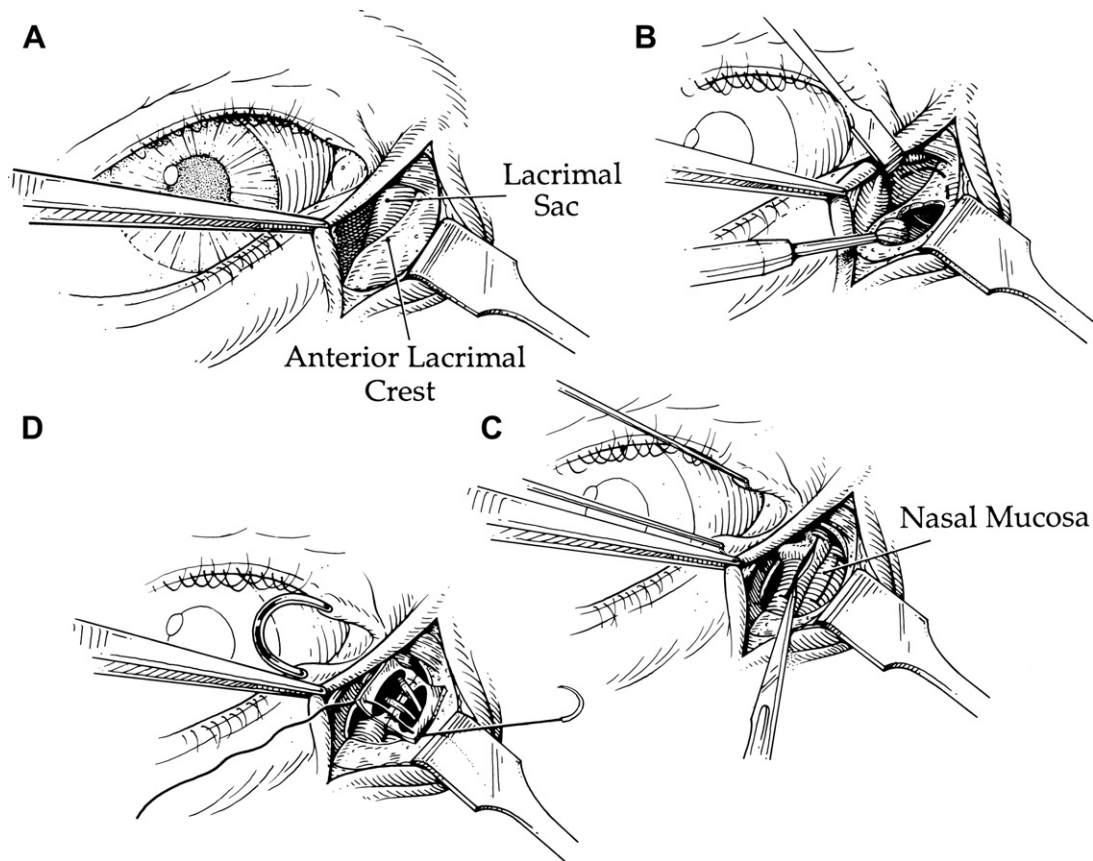


Fig. 34 Technique for DCR. (A) Exposure of lacrimal sac and anterior lacrimal crest. (B) Bony ostium made through lacrimal bone and anterior lacrimal crest. (C) Lacrimal sac and nasal mucosa incised longitudinally. (D) Anterior and posterior lacrimal sac and nasal flaps sutured together. Long-term lacrimal stents passing from the inferior and superior canaliculi into the nasal cavity are utilized for best results. (From Salin MB, Smith BM. Diagnosis and treatment of midface fractures. In: Fonseca RJ, editor. Oral and maxillofacial trauma. vol. 2. St Louis (MO): Elsevier; 2005. p. 685; with permission.)

Frontal sinus fractures

The frontal sinus is intimately associated with the midface via its communication with the NOE region. Because of this relationship, the management of frontal sinus fractures is included in this article. Frontal sinus fractures rarely occur in isolation. Approximately 70% are associated with other maxillofacial injuries. The most common cause of injury is motor vehicle collisions. Associated neurologic injuries present in greater than 50% of patients, and approximately 25% present with an ophthalmologic injury. A subdural or epidural hematoma requiring emergency surgical intervention occurs in 8% to 10% of patients with frontal sinus fractures.

Embryology and anatomy

The nasal and frontal bones begin intramembranous ossification around 50 days of gestation, with signs of frontal sinus development beginning around 4 months in utero. Pneumatization of the frontal sinus is highly variable, with up to 4% of the population having no discernable sinus present. The sinus reaches its adult size in the late teens and has an average volume of 5 to 16 mL. The average height of the sinus is 32 mm, and the average width is 26 mm. The frontal sinus is bordered superiorly by the frontal bone. The inferolateral aspect is adjacent to the supraorbital rim and orbital roof. Anterior and inferior are the nasofrontal ducts, which are confluent with the

nasoethmoidal complex. The posterior wall borders the intracranial cavity and skull base and is tightly adherent to the dura.

The frontal sinus protects the brain and intracranial contents by serving as a shock absorber. Normal sinus function involves mucus clearance via pseudostratified columnar respiratory epithelium from the sinus, through the nasofrontal outflow tract (nasofrontal duct), with drainage into the nose inferior to the middle turbinate. Any obstruction to normal drainage can lead to a mucocoele or infection, with resultant erosion into the cranial vault or orbit.

Diagnosis

Initial evaluation of the patient with a frontal sinus fracture should follow the workup described earlier for midface trauma. Particular attention should be paid to intracranial injury. Specific clinical findings may include a palpable bony step, a contour deformity, crepitus, mobility of bony segments, and paresthesia of the forehead and scalp. Close attention should also be paid to the NOE region for injuries that may contribute to sinus outflow obstruction. Rhinorrhea, when present, may indicate a CSF leak. The halo test can be used to confirm the presence of CSF. When the fluid is placed on a piece of filter paper, a ring of clear CSF surrounds a central component of blood. Alternatively, the fluid can be tested for glucose and chloride to differentiate between serum and nasal

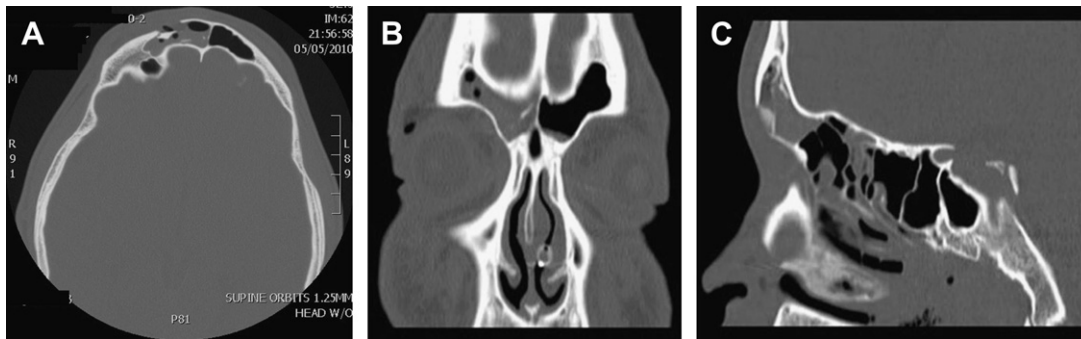


Fig. 35 CT scan of patient with fracture of right anterior table of frontal sinus: (A) axial view, (B) coronal view, and (C) sagittal view.

secretions. The most accurate confirmation is made by testing the fluid for β_2 transferrin. This laboratory test can take up to 4 days to process and can therefore cause an unwanted delay in diagnosis and treatment.

Imaging via CT in axial, coronal, and sagittal planes is essential to the diagnosis of specific fracture elements (Fig. 35). Axial slices best evaluate the anterior and posterior tables of the sinus. Coronal slices are useful in the evaluation of the sinus floor and orbital roof, and sagittal slices provide valuable information about the nasofrontal outflow tracts.

Treatment and management

The primary goal of frontal sinus fracture management is to restore form and function and minimize morbidity and complications. Management can be divided into 2 main categories: nonoperative observation and surgical intervention. Several investigators have suggested specific treatment algorithms based on criteria such as the location of the fracture, the degree of bony displacement, and the presence of nasofrontal outflow obstruction (Figs. 36 and 37). These algorithms are based primarily on clinical judgment, surgical experience, and knowledge of sinus pathophysiology and the complications that may occur after frontal sinus injury. The fact remains that there is a lack of long-term data in the literature and there continues to be debate on this subject. In general, most investigators promote nonsurgical management in cases involving minimal displacement of the anterior and posterior tables with an intact nasofrontal outflow tract. These patients should be followed at regular intervals for early detection of complications. They should be placed on sinonasal precautions,

and the use of decongestants is recommended in the first weeks after injury.

Surgical management of frontal sinus fractures is reserved for cases involving a greater degree of bony displacement or damage to the frontonasal outflow tract. Those cases involving a significant frontobasilar injury with a persistent CSF leak or comminuted fractures of the region surrounding the nasofrontal outflow tract are of particular concern. Surgical management may involve any or several of the following:

- Anterior table reconstruction
- Nasofrontal outflow tract management
- Sinus obliteration
- Cranialization

Surgical access can be achieved in several ways. The most common approach is the coronal flap. This approach provides excellent exposure of the frontal bone and NOE region. It also allows for a pericranial flap to be developed and split-thickness calvarium to be harvested, if necessary. Balding patients or those with a receding hairline should have the incision placed more posteriorly, to avoid a noticeable scar. Existing lacerations may also be used if broad enough exposure will be accomplished. Other options include the direct, open-sky, and gullwing approaches. These approaches should generally be avoided to prevent the unsightly scars that they typically produce.

Anterior table reconstruction

Displaced anterior table fractures without involvement of the nasofrontal outflow tract can be anatomically reduced and

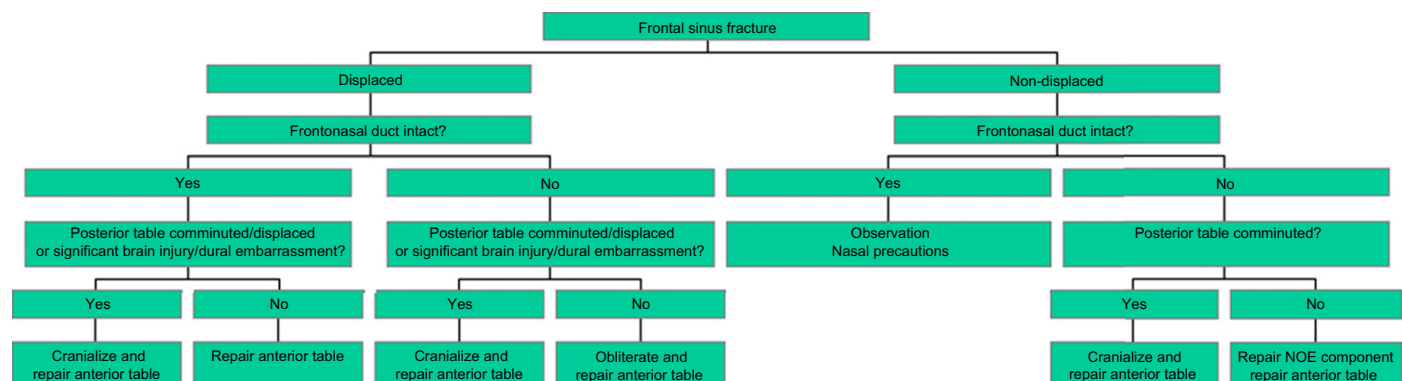


Fig. 36 Algorithm for repair of frontal sinus fractures. (From Bell RB, Dierks EJ, Brar P, et al. A protocol for the management of frontal sinus fractures emphasizing sinus preservation. J Oral Maxillofac Surg 2007;65:825; with permission.)

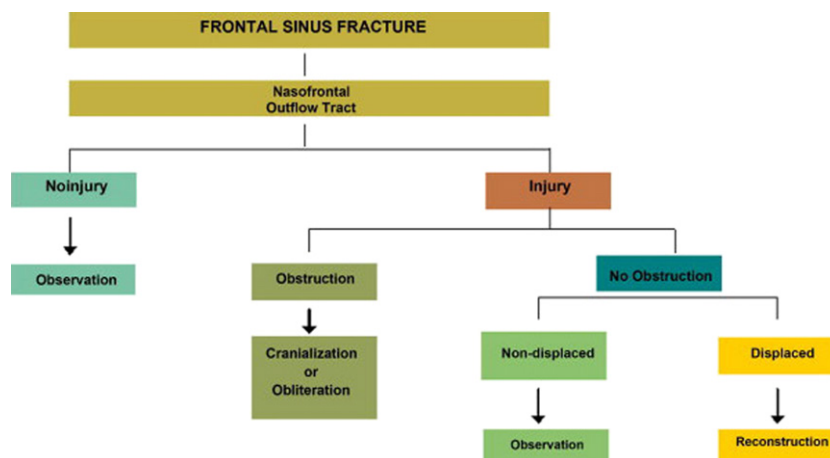


Fig. 37 Frontal sinus fracture treatment algorithm. (From Stanwix MG, Nam AJ, Manson PN, et al. Critical computed tomography diagnostic criteria for frontal sinus fractures. *J Oral Maxillofac Surg* 2010;68:2714; with permission.)

fixed with titanium mesh or titanium/resorbable plates and screws (Fig. 38). Although there is no consensus, removal of the sinus membrane and obliteration of the sinus are not believed to be necessary in these cases. Anatomic reduction of anterior table fractures prevents contour deformities that are unacceptable to the patient. Simple methods of reduction include the end of a periosteal elevator or a Carroll-Girard screw. If bone has been lost, autogenous bone grafts can be used as necessary. In addition to this open technique, endoscopic approaches are beginning to gain popularity with many surgeons.

Obliteration

When there is damage to the nasofrontal and NOE region, an attempt can be made to assess the patency of the nasofrontal outflow tract from above. Propofol is injected into the outflow tract, and its presence in the nose inferior to the middle turbinate is assessed. If drainage is poor or absent, sinus obliteration should be considered. Sinus obliteration involves the following (Fig. 39):

- Complete removal of sinus mucosa
- Permanent occlusion of the nasofrontal outflow tract
- Obliteration of dead space

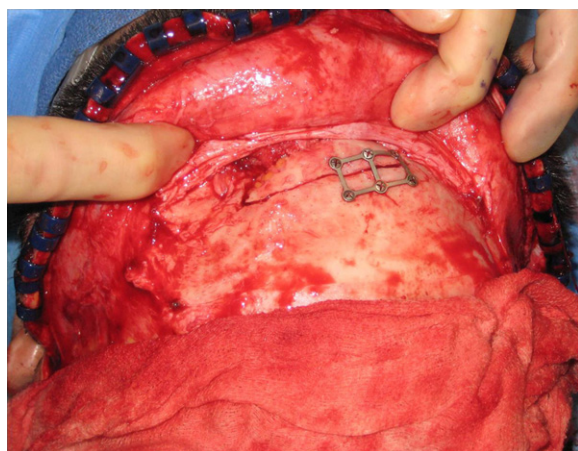


Fig. 38 Repair of anterior table of frontal sinus.

After surgical access, the anterior table is removed. The sinus mucosa is then meticulously removed with curettes or rotary instruments (see Fig. 39B). The nasofrontal outflow tract can then be occluded (see Fig. 39C). A commonly used technique involves the use of fibrin glue and temporal fascia. A pedicled pericranial flap can also be used for this purpose. The sinus is filled with an autologous material (see Fig. 39F). Common fillers include abdominal fat, temporalis muscle, and autologous bone. The anterior table is then reconstructed, as described earlier (see Fig. 39I).

Cranialization

Frontal sinus fractures involving significant displacement or comminution of the posterior table often require cranialization to prevent devastating complications such as meningitis and mucopyoceles. Any injury in which a suspicion of intracranial involvement is present warrants neurosurgical consultation. If there is a significant frontobasilar injury with a persistent CSF leak, exploration of the cranial base and dural repair are likely necessary. Cranialization is typically performed after a bifrontal craniotomy is performed by a neurosurgeon. With the dura retracted and protected, the posterior table is removed with a rotary or hand instrument (Fig. 40). The sinus mucosa is then removed, the nasofrontal outflow tract is occluded, and a pericranial flap is used to separate the aerodigestive tract from the intracranial cavity (Fig. 41). The anterior table is reconstructed, as described earlier.

Postoperative management

In the immediate postoperative period, a CT scan should be taken to evaluate the reconstruction and to acquire a baseline for comparison for future studies. Postoperative pain is controlled as necessary. Antibiotics should be used during the perioperative period to prevent infection. If the initial trauma created a contaminated wound, we recommend extending the antibiotic course for 7 to 14 days postoperatively. As discussed earlier, decongestants such as pseudoephedrine and oxymetazoline spray should be used in the postoperative period to maintain sinus patency for cases not involving occlusion of the nasofrontal outflow tract.

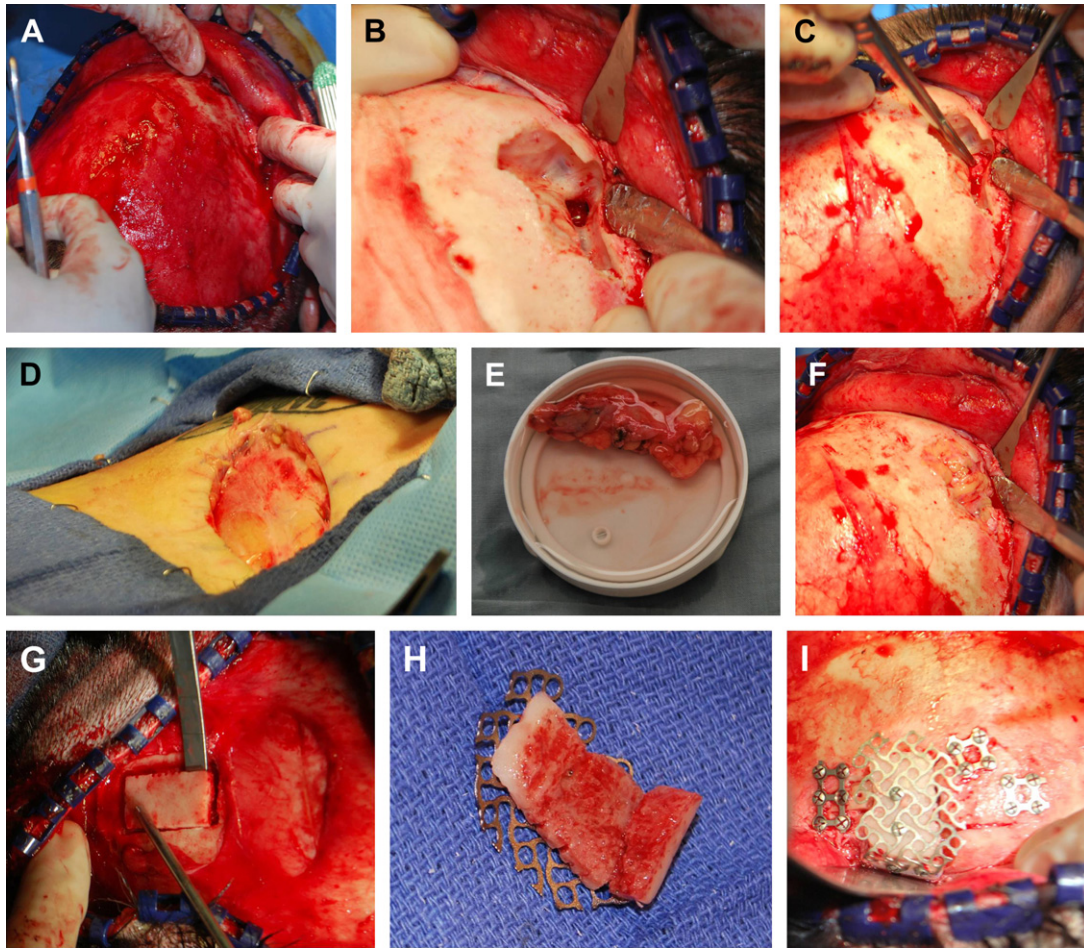


Fig. 39 Obliteration of the frontal sinus. (A) A coronal flap elevated in patient with anterior table fracture and damage to the right nasofrontal duct. (B) The anterior table has been removed, and the sinus mucosa has been debrided. The nasofrontal duct is visible at the floor of the sinus. (C) The nasofrontal duct is occluded with temporal fascia. (D, E) Harvesting of abdominal fat. (F) The sinus filled with the abdominal fat. (G, H) Harvesting of split-thickness calvarium for reconstruction of anterior table. (I) Reconstruction of anterior table.

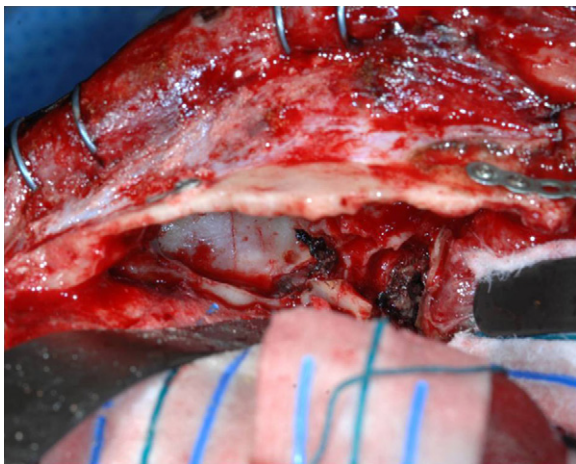


Fig. 40 Cranialization of frontal sinus. The posterior table of the frontal sinus has been removed. With the brain retracted, the nasofrontal ducts are visualized at the floor of the sinus.



Fig. 41 A pedicled pericranial flap can be used to separate the aerodigestive tract from the intracranial contents when cranializing the frontal sinus.

Long-term serial follow-up examinations are critical for these patients, because of the devastating late complications that can occur. We recommend the following strategy:

- Weekly up to 1 month
- Every 3 months up to 1 year
- Every year up to 5 years
- Every 5 years indefinitely

Complications

Early and late complications may occur after the surgical management of frontal sinus fractures. Early complications include pain, headaches, CSF rhinorrhea, sinusitis, meningitis, brain abscess, osteomyelitis, residual aerodigestive communication, pneumocephalus, and contour irregularities. Late complications may include mucocoeles and mucopyoceles.

CSF rhinorrhea

Tears in the dura may lead to CSF leaks. These tears may be iatrogenic in nature or may be residual unrepaired tears from the inciting trauma. After the nasal fluid is confirmed to be CSF via the methods described earlier, this condition can be managed by conservative measures (elevation of the head, sinus precautions), lumbar diversion, intracranial surgical repair, extracranial surgical repair, and transnasal endoscopic repair.

Mucocoeles/mucopyoceles

Mucocoeles are expansile cysts lined with respiratory ciliated epithelium. Mucocoeles may occur after frontal sinus trauma or reconstruction if residual epithelium becomes trapped in fracture segments or if the normal sinus drainage is altered because of obstruction of the nasofrontal outflow tract. Mucocoeles can be locally destructive and can erode through the posterior table and extend intracranially. Infected mucocoeles are called mucopyoceles. Treatment consists of surgical removal or marsupialization via an external or endoscopic approach. Mucopyoceles require prompt surgical treatment and administration of intravenous antibiotics.

Summary

The management of midface trauma continues to challenge maxillofacial surgeons. The complex local anatomy and functional and cosmetic importance of the region make precise surgical correction and reconstruction essential to success.

Further readings

Bell RB, Dierks EJ, Homer L, et al. Management of cerebrospinal fluid leak associated with craniomaxillofacial trauma. *J Oral Maxillofac Surg* 2004;62:676.

Bell RB. Management of frontal sinus fractures. *Oral Maxillofac Surg Clin North Am* 2009;21:227.

Doonquah L, Brown P, Mullings W. Management of frontal sinus fractures. *Oral Maxillofac Surg Clin North Am* 2012;24:265.

Ellis E, el-Attar A, Moos KF. An analysis of 2,067 cases of zygomatico-orbital fracture. *J Oral Maxillofac Surg* 1985;43:417.

Ellis E. Fractures of the zygomatic complex and arch. In: Fonseca RJ, editor. *Oral and maxillofacial trauma*, vol. 2. St Louis (MO): WB Saunders; 2005.

Ellis E. Sequencing treatment for naso-orbito-ethmoid fractures. *J Oral Maxillofac Surg* 1993;51(5):543–58.

Fabio R, Paolo B, Valeria G, et al. Role of the maxillofacial surgeon in the management of severe ocular injuries after maxillofacial fractures. *J Emerg Trauma Shock* 2011;4(2):188–93.

Fattahi T, DiPasquale J. Utility of the pericranial flap in frontal sinus and anterior cranial fossa trauma. *Int J Oral Maxillofac Surg* 2009;38:1263.

Follmar KE, DeBruijn M, Baccarani A, et al. Concomitant injuries in patients with panfacial fractures. *J Trauma* 2007;63:831.

He D, Blomquist PH, Ellis E. Association between ocular injuries and internal orbital fractures. *J Oral Maxillofac Surg* 2007;65(4):713–20.

Hendrickson M, Clark N, Manson PN, et al. Palatal fractures: classification, patterns, and treatment with rigid internal fixation. *Plast Reconstr Surg* 1998;101:319.

Knight JS, North JF. The classification of malar fractures: an analysis of displacement as a guide to treatment. *Br J Plast Surg* 1961;13:325.

Lee SS, Huang SH, Wu SH, et al. A review of intraoperative airway management for midface facial bone fracture patients. *Ann Plast Surg* 2009;63:162.

LeFort R. Etude expérimentale sur les fractures de la machoire supérieure [Experimental study of fractures of the upper jaw parts I and II]. *Rev Chir Paris* 1901;23:208 [Original in French; translation by Tessier P published 1972].

McRae M, Frodel J. Midface fractures. *Facial Plast Surg* 2000;16:107.

Park SP, Kim YJ, Kim H, et al. Prevalence of diplopia and extraocular movement limitation according to the location of isolated pure blowout fractures. *Arch Plast Surg* 2012;39:204–8.

Perino KE, Zide MF, Kinnebrew MC. Late treatment of malunited malar fractures. *J Oral Maxillofac Surg* 1984;42:20.

Potter JK, Ellis E. Biomaterials for reconstruction of the internal orbit. *J Oral Maxillofac Surg* 2007;62(10):1280–97.

Rafael B, Ravit B, Eran R, et al. Neurosensory changes in the infraorbital nerve following zygomatic fractures. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2005;99:657.

Salin MB, Smith BM. Diagnosis and treatment of midface fractures. In: Fonseca RJ, editor. *Oral and maxillofacial trauma*, vol. 2. St Louis (MO): Elsevier; 2005.

Simmons O, Manson PN. Endoscopic management of orbital and frontal sinus fractures. *Craniomaxillofac Trauma Reconstr* 2009;2:177.

Stanwix MG, Nam AJ, Manson PN, et al. Critical computed tomographic diagnostic criteria for frontal sinus fractures. *J Oral Maxillofac Surg* 2010;68:2714.

Zingg M, Laedrach K, Chen J, et al. Classification and treatment of zygomatic fractures: a review of 1,025 cases. *J Oral Maxillofac Surg* 1992;50:778.

Ocular Injuries, Triage, and Management in Maxillofacial Trauma

Jeffrey P. Blice, MD, CAPT, MC, USN ^{a,b}

KEYWORDS

• Triage • Eye trauma • Ruptured globe • Ocular examination

KEY POINTS

- Key historical information for the triage of ocular trauma includes the mechanism and nature of the injury, the presence of eyewear at the time of the injury, and the status of vision before injury.
- Examination findings indicative of a ruptured eye are poor vision (hand motions or less); extensive subconjunctival hemorrhage; poor ocular motility in all directions; a large hyphema; or intraocular contents visible through a wound.
- Visual acuity is the most important prognostic indicator after eye trauma and needs to be measured and recorded in a reliable way.
- Findings of a ruptured eye require immediate consultation with an ophthalmologist.

Introduction

The most important thing one can do for a patient with facial trauma is correctly identify a concurrent severe eye injury for appropriate and timely referral to an ophthalmologist. This article provides practical information allowing one to make intelligent decisions with regard to ocular injuries in the setting of simple or complex facial injuries. Some eye injuries may not require emergency care by an ophthalmologist but can be managed initially without urgent consultation. Other injuries need urgent referral for evaluation and treatment. The goal of the surgeon or provider is to appropriately discriminate between the two. Accumulating the appropriate information and accurately presenting that information to the ophthalmologist triggers an appropriate response.

History and physical examination of the patient with the traumatized eye

An accurate history of how the injury occurred is the first important piece of information to gather [Box 1](#). The nature of the injury can raise the suspicion of a more severe injury.

Disclaimer: The views expressed in this article are those of the author and do not necessarily reflect the official policy or position of the Department of the Navy, Army, Department of Defense, or the US Government. I certify that all individuals who qualify as authors have been listed; each has participated in the conception and design of this work, the analysis of data (when applicable), the writing of the document, and the approval of the submission of this version; that the document represents valid work; that if we used information derived from another source, we obtained all necessary approvals to use it and made appropriate acknowledgments in the document; and that each takes public responsibility for it.

^a Ophthalmology, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, MD 20889, USA

^b Uniformed Services University of Health Sciences, Bethesda, MD, USA
E-mail address: jpblice@gmail.com

Mechanisms involving high-velocity small projectiles are more concerning for penetrating ocular injuries. Typical histories in these cases involve breaking glass from bottles used as weapons, explosions of any kind, or metal-on-metal contact during industrial or tool use. Blunt-force injuries also can cause serious ocular trauma. Beatings with an object or edge small enough to fit into the opening between the orbital rims are more likely to cause direct ocular trauma. Explosive force in the absence of projectiles can rupture sclera or damage intraocular structures; a determination of the distance from an explosion and a general assessment of its power are important in building the case for a severely injured eye.

Another useful historical detail is whether or not eyeglasses were worn at the time of the injury. A completely intact set of eyeglasses surviving trauma suggests the area of the orbit has been spared, and the eye spared with it. Conversely, a completely destroyed pair of eyeglasses with shattered or damaged lenses suggests enough destructive force to cause severe injury to the eye and surrounding tissue. The use of contact lenses at the time of the injury, the nature of those lenses (hard or soft), and the status of them after the injury are useful bits of information when evaluating the patient and discussing the case with an ophthalmologist. Historical confirmation that an eye could see and read before trauma is a critical piece of information. An eye that did not see before an injury does not see after the event.

Visual acuity

Measuring how well an injured eye can see is the first and most important step to determine the urgency of an ophthalmic evaluation. A well-determined visual acuity is a critical branch point in the decision path of ocular triage and the most important prognostic indicator in eye trauma. Poor vision equals damage to the anatomic structure of the eye or at a minimum introduction of opacities in the otherwise optically transparent ocular structures. Either way, this is when the ophthalmologist needs to sort out the problem [Box 2](#).

Box 1. Critical historical information

- Mechanism and nature of the injury
- Eyewear at the time of the injury
- Status of vision before injury

An everyday visual acuity measurement is usually recorded in an eye professional's office using a standard Snellen acuity chart. Acuity measurements using a standard chart are recorded from 20/400 to 20/20. The numerator of these fractions refers to the normal test distance of 20 ft. The denominator refers to the line on the chart that a "normal" patient should be able to see at 20 ft. In patients who are unable to see the eye chart adequately enough to read it, other approximations are recorded, such as "counts fingers at 5 feet," "hand motion at 3 feet," or "light perception."

To a professional who does not routinely record visual acuity, the standard methods are likely not available. However, other methods are equally important and need to be pursued. Newspapers, magazines, and sweetener packets can be used as an estimation of acuity. The goal is to determine the best acuity possible in a potentially severely injured eye. This can be done with eyeglasses at reading distance, 14 in, or at 10 ft; however, if vision is actually better without eyeglasses it should be recorded without. However the vision is measured, the method needs to be recorded with it. Examples of appropriately recorded acuity measurements include the following:

"Patient is able to read with right and left eye small print on Splenda packet at about 14 in with reading eyeglasses."

"Patient can read Washington Post story print at 14 in without any eyeglasses."

"Patient can only accurately count fingers placed 2 ft in front of left eye with or without eyeglasses."

"Patient can only perceive light or dark with the right eye using otoscope light at 6 in."

A magnified view

If one is not an eye care professional one will not have the "right" equipment to examine an eye. A slit lamp is a biomicroscope, magnification with a light source. A maxillofacial surgeon is likely to have a pair of surgical loupes to assist in examination. Magnification is the first assistance in examining the eye. Use what is available to get a magnified view; a handheld magnifying lens is better than nothing. A pair of magnifying surgical loupes is better. Light is needed to see. Get a good source of light on the eye; brighter is better. Light the eye from the front, then the side. Different angles provide different information. Are the eyelids in the way? They need to

be moved. One drop of sterile ophthalmic anesthetic to permit an examination can transform a most uncooperative patient into the most cooperative one. Retracting the lids should be done carefully. Only apply pressure on the orbital rim to retract lid tissue. Lid retractors can also be used carefully but risk the opportunity to apply pressure to the eye itself. This is usually best left for the ophthalmologist. Examining the eye, even for purposes of triage, is a skill. Practice makes perfect or at least improvement.

Clinical examination indicators of a severely injured eye

One does not have to be an ophthalmologist to recognize the pertinent examination findings of an eye that requires the attention of an ophthalmologist. However, there is a need to recognize the following key findings that distinguish a routine consultation from an emergent or urgent one.

Poor vision after facial trauma with a history suspicious for an ocular injury is probably enough to warrant an emergent evaluation by an ophthalmologist. What does "poor vision" mean? This is why an accurate measurement of acuity in the context of an accurate history is critical. An injured pilot, who by definition should have excellent vision in each eye, with loss of vision to 20/400 in one eye is an emergency. A patient with 20/80 vision who wore a patch for a lazy eye on the same side as a facial injury is not necessarily an urgent evaluation. Consultation with the ophthalmologist helps sort out the urgency of an evaluation in cases, but having the right data collected makes that discussion easier.

Subconjunctival hemorrhage is a common occurrence spontaneously outside of a traumatic event. These asymptomatic thin or small spontaneous hemorrhages are themselves harmless. A worrisome subconjunctival hemorrhage in the setting of trauma is extensive, often surrounding the cornea for 360 degrees. The hemorrhage is large, heaping up the conjunctiva enough that in some cases the lids do not close completely or are elevated off the surface of the cornea. A hemorrhage this large implies a rupture of the globe itself. The bleeding originates from the choroid beneath the sclera; in addition, liquid intraocular contents may be accumulating in the same space as the hemorrhage. A small subconjunctival hemorrhage is less worrisome in the setting of trauma unless the history is suspicious for a projectile injury and a penetrating foreign body [Box 3](#).

Poor motility in all directions of gaze is another very worrisome finding in facial trauma. Inability to infraduct or supraduct an eye may indicate a trapped and injured inferior rectus muscle. Lateral and medial movement are preserved. However, poor motility in all directions is an indication of a collapsed eye. The extraocular muscles have a poor mechanical advantage on a soft and misshapen globe. It is

Box 2. Important ocular physical examination elements

- Visual acuity
- Lids
- Conjunctiva
- Cornea
- Iris and pupil

Box 3. Examination findings suspicious of a ruptured globe

- Very poor vision (hand motions or less)
- 360 degrees of subconjunctival hemorrhage
- Poor ocular motility in all directions
- Large hyphema
- Visible intraocular contents through a wound

unlikely that such an injury would occur without a large subconjunctival hemorrhage, but if noted on examination it should be discussed with the ophthalmologist.

Gross anatomic derangement of important ocular structures is a clear indication for an emergent evaluation by an ophthalmologist. Visible corneal wounds, intraocular contents prolapsing through a wound, missing sectors of iris, loss of corneal clarity, and lens material or blood layered in the anterior chamber are all conditions warranting emergency evaluation. Although surgeons may be comfortable with the repair of full-thickness lacerations, lid lacerations involving the puncta of either the upper or lower lid warrant consultation with an ophthalmologist or oculoplastic subspecialist.

Other indications of severe ocular trauma are important, but from a practical standpoint require examination skills or equipment not likely to be at the disposal of the maxillofacial surgeon. An afferent pupillary defect is a reliable indicator of damage to the eye or optic nerve. Reliable detection of this pupillary abnormality is unlikely unless specially trained and practiced in the examination for it. Vitreous hemorrhage is also a reliable indicator of severe injury, but without experience in examining intraocular structures or skilled use of special examination equipment accurate detection is unlikely. Indirect detection of vitreous hemorrhage by assessment or visual acuity is a better screening tool for the maxillofacial surgeon.

Radiographic imaging

The maxillofacial surgeon is well acquainted with the value facial computed tomography (CT) has in the management of facial trauma. In addition to the bone abnormalities seen in trauma there are characteristic findings that point to severe intraocular involvement. Attending to the soft tissues in the orbit can provide information to support a suspected diagnosis or raise the suspicion of an ocular injury requiring closer scrutiny.

Intraocular air on CT is conclusive evidence that the integrity of the eye wall has been violated. Usually a violation violent enough to trap air in the eye does not escape detection by clinical examination. Regardless of other findings on examination the presence of intraocular air on CT warrants immediate referral to an ophthalmologist. Air trapped under the lid can occur in the absence of trauma and needs to be correctly identified as such to avoid confusion.

An intraocular foreign body is conclusive finding on CT that the eye wall has been violated (Figs. 1 and 2). CT protocols with thick overlapping sections are unlikely to miss a foreign body. Metal, metal containing glass, or mineral objects appear relatively radiodense and are easy to detect. Fresh vegetable matter and plain glass or sand may appear close to the density of water. Dry wood has the radiodensity of air. A large foreign body likely causes a rupture with clinical findings of poor vision and large subconjunctival hemorrhage. However, a very small sharp foreign body can penetrate the eye cleanly with minimal damage and bleeding; vision immediately afterward can be excellent. A history with the potential for small high-velocity projectiles makes this more likely. Regardless of other findings on examination the presence of an intraocular foreign body on CT warrants immediate referral to an ophthalmologist.

Abnormal ocular contour or a squared-off globe is another worrisome finding on CT (Fig. 3). An eye with loss of contents or open wound may no longer maintain the spherical shape of a normal eye. The presence of this finding is unlikely in the absence of extremely poor vision and significant subconjunctival



Fig. 1 Axial CT of patient with a large intraocular foreign body. Fox shield is seen in place over injured eye.

hemorrhage. If noted on CT, however, an ophthalmic evaluation is warranted unless the clinical examination findings are convincingly normal.

Entrapment of an extraocular muscle in an orbital fracture is often stated as a reason for emergent evaluation by an ophthalmologist (Fig. 4). However, the clinical examination of ocular motility is a much more reliable indicator of true muscle entrapment. Unless there is evidence of direct ocular injury requiring emergent evaluation, the ophthalmologist may view a full ocular examination as less urgent. Surgical repair of the fracture may still be indicated in the absence of double vision if the fracture is large and more than 2 mm of enophthalmos is present.

What does a magnified normal eye look like?

To recognize abnormalities after trauma, one must be familiar with the anatomy and appearance of the normal eye. Eyelid skin is no different than other skin on the body except it is hairless and very thin. The lid margin should rest against the

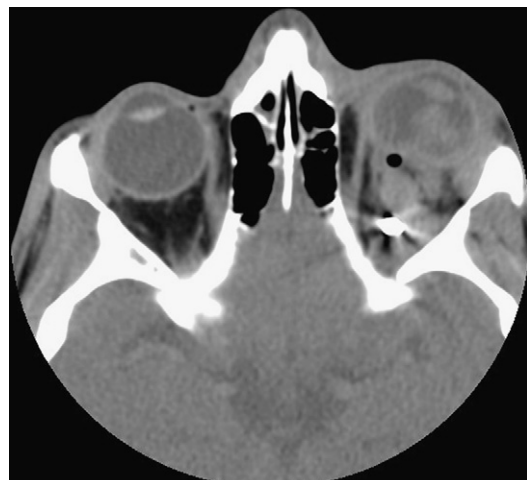


Fig. 2 Axial CT demonstrating large intraorbital foreign body. Air is present behind globe suggesting posterior globe rupture.



Fig. 3 Coronal CT of orbital floor fracture. Prolapsed orbital contents are visible. The inferior rectus appears directly involved. Examination confirmed a severe limitation in infraduction of the right eye.

globe with lashes oriented outward. The puncta of each upper and lower lid should be rotated slightly in toward the globe to remain in contact with the tear lake. The conjunctiva is normally vascularized and transparent so that the connective tissue surrounding the globe and the white nonvascular sclera are visible. The cornea is clear. Iris should be easily visible through the cornea. The pupil is round and symmetric in a normal eye. The lens is normally clear and unless the pupil is dilated may be difficult to discern as a separate structure without a slit lamp (Fig. 5).

Lid injuries

Lids can be contused, abraded, lacerated, or avulsed like any other tissue. A maxillofacial surgeon should be able to recognize and manage these traumatic injuries. Full-thickness lid lacerations that can be repaired by surgeons other than ophthalmologists should always raise the suspicion of a more



Fig. 4 Axial CT of severely damaged right globe. Note flattened contour. Small intraocular foreign body is seen in the left eye laterally.

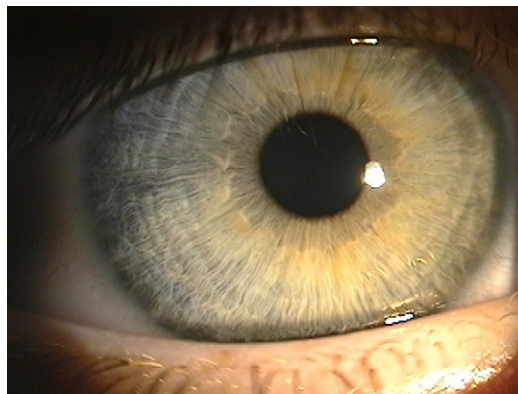


Fig. 5 Magnified view of normal eye.

serious ocular injury. The same mechanism causing a full-thickness laceration can cause a scleral or corneal laceration. In the presence of normal vision and normal examination of the external ocular structures an urgent ophthalmic consultation may only delay a primary closure [Box 4](#).

Lacerations that involve a lid margins can be managed by the maxillofacial surgeon. However, repair of lacerations that involve the lacrimal puncta or canaliculus are more involved and require probing and stenting. A laceration near the medial canthus needs to be explored aggressively. Distracting the lids to confirm a laceration is small and does not involve the puncta is essential. Those lacerations that are "too close to call" (Fig. 6) should be evaluated urgently by an ophthalmologist.

Conjunctival injuries

Isolated conjunctiva injuries are likely to be associated with minor trauma. A typical history usually involves a finger poke in the eye or glancing injury with an object. Vision should be near normal and other ocular structures should look normal. However, the pain associated with them can be significant. Application of an ophthalmic topical anesthetic can aid in the appropriate examination of the patient so an accurate acuity and external examination can be performed. Because the conjunctiva is relatively transparent the edge of the laceration is often marked by an area of thin subconjunctival hemorrhage (Fig. 7). A conjunctival laceration can be allowed to heal by secondary intention but heals faster if sutured closed. Emergent repair is not required, but best planned in consultation with the ophthalmologist.

A history of an explosive or high-velocity projectile injury in association with a conjunctival laceration (Fig. 8) should raise the suspicion of a serious injury. Consultation with an ophthalmologist to rule out an intraocular foreign body is

Box 4. Urgent ophthalmology consult indicated

- Examination findings supporting globe rupture
- Examination findings supporting an intraocular foreign body
- Hyphema
- Disordered ocular anatomy
- Poor vision after trauma
- Lid margin lacerations



Fig. 6 Lid laceration of the upper lid that is either dangerously close to or involving the lacrimal drainage system.

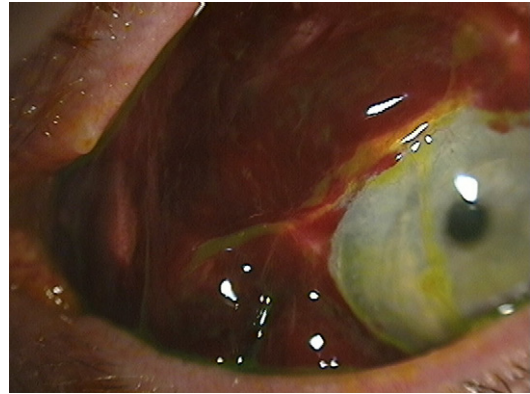


Fig. 9 Thick massive subconjunctival hemorrhage indicating a ruptured globe.



Fig. 7 Conjunctival laceration medial edge retracted with thin hemorrhage. Underlying tissue is normal.

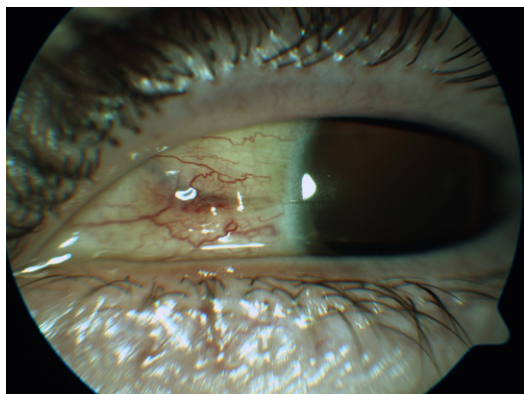


Fig. 8 Conjunctival laceration with visible "L"-shaped scleral rupture beneath from sharp projectile.

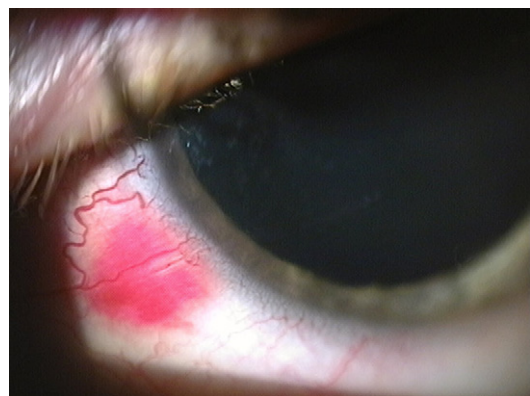


Fig. 10 Small thin subconjunctival hemorrhage without findings of other anatomic abnormalities. This is unlikely to represent a severe injury.

required. Vision can be normal, hemorrhage can be minimal, and motility normal. If a CT scan is obtained, the foreign body may be visible on CT. This is the unusual circumstance when a severely injured eye may not look severely injured.

The importance of subconjunctival hemorrhage as an examination finding in a severely injured eye has been discussed previously. A large severe hemorrhage as pictured in [Fig. 9](#) indicates a severely injured eye and likely is associated with poor vision and poor motility. The typical subconjunctival hemorrhage shown in [Fig. 10](#), however, is small, thin, and normal anatomy. Normal vision and normal motility is the rule. Spontaneous resolution is expected even in cases without a history of trauma.

Corneal injuries

The cornea is normally clear, smooth, and domed. Any opacity in the cornea, significant irregularity, or contour change is a good indication of an injury. Most commonly the cornea is merely abraded. The thin epithelial layer overlying the collagenous skeleton of the cornea is removed ([Fig. 11](#)). Vision can be normal or slightly diminished. A pronounced foreign body sensation is typical. A small subconjunctival hemorrhage may accompany the abrasion. Motility is normal and the anatomy of the eye otherwise appears normal. An abrasion heals in 24 to 48 hours left untreated or treated merely with a lubricating antibiotic ophthalmic ointment. Although fluorescein staining

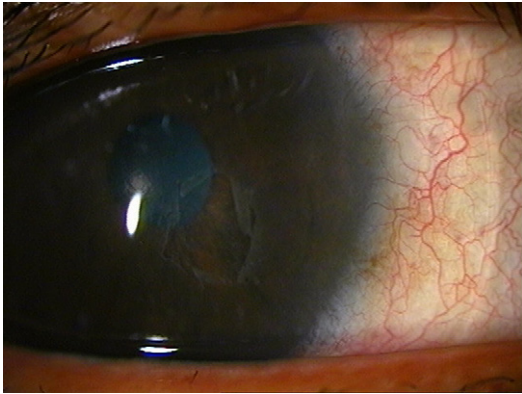


Fig. 11 Superficial corneal abrasion (epithelial defect).

used with a blue light is useful to confirm the finding, it is not required or may not be readily available.

Corneal lacerations and ruptures are much more serious injuries requiring emergent evaluation and treatment by an ophthalmologist. Large injuries are usually accompanied by the usual indicators of a severe injury: poor vision, visible intra-ocular contents, and massive subconjunctival hemorrhage. Small lacerations or ruptures may be self-sealing with good vision. These are usually in the setting of injury with a small sharp object or projectile.

Iris injuries

An easily visible indication of severe ocular injury is distortion of the normal pupil shape. A small corneal laceration or rupture can be sealed with iris tissue. This irregular shape or tear-drop shape needs to be examined to ensure no significant injury has occurred (Fig. 12). Blunt injury to the iris can cause iris sphincter disruption or even more significantly a disinsertion of the iris from the normal origin called an iridodialysis (Fig. 13). Traumatic pupil dilation with poor light reaction or a large flattened edge creating a "D" shape to the normally "O"-shaped pupil indicates significant trauma. Any injury severe enough to cause an abnormal appearance to the iris and pupil warrants consultation with an ophthalmologist and likely emergent evaluation. The severest of these injuries is usually accompanied by other findings outlined previously.

A hyphema is blood from disruption of the iris vasculature present in the normal space, the anterior chamber, between the

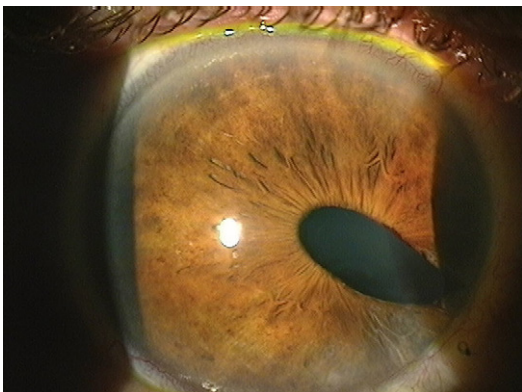


Fig. 12 Pupil distortion indicating a severe blunt injury, rupture, or laceration of the cornea.

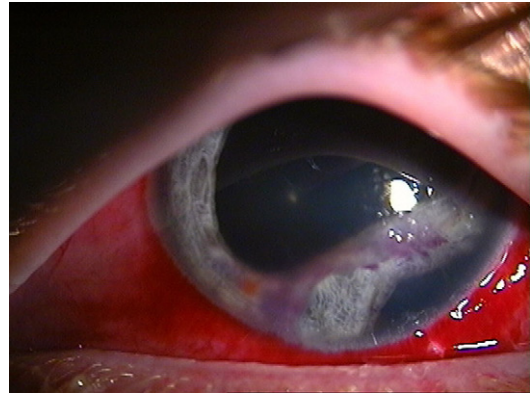


Fig. 13 Iridodialysis with moderate subconjunctival hemorrhage and corneal epithelium irregularity, from paintball trauma.

iris and the cornea. Hyphema equates with severe iris trauma. If bleeding is severe enough the entire chamber can be filled (Fig. 14). The blood can be so dense that the cornea appears black; no iris at all can be seen. This appearance has been given the moniker "8-ball hyphema" (Figs. 15 and 16). Vision is markedly poor, usually "hand motion" or "light perception"; subconjunctival hemorrhage is usually present to some degree. Motility is usually preserved if there is no simultaneous rupture.

A large layered hyphema is visible without magnification; a small layered hyphema can be harder to detect without magnification but equally important. Even a small hyphema usually causes a significant noticeable decrease in acuity. The layer of blood tends to be inferior or toward the lowest point of the anterior chamber (Fig. 17).

Blood may not be the only substance seen in the anterior chamber. An injury serious enough to cause a hyphema can also cause disruption of lens. Although normally clear, when traumatized, lens proteins become opaque and can be seen as white material. In this case vision is significantly diminished from lens opacification or traumatic cataract. Any blood or lens material present in the anterior chamber warrants emergent evaluation and treatment by an ophthalmologist (Fig. 18).

Large globe ruptures with disrupted normal anatomy

The most severely injured eyes are easiest to spot; however, they also have the worst prognosis. Large scleral or corneal



Fig. 14 Total severe hyphema. Blood entirely filling the anterior chamber.



Fig. 15 Facial and brow lacerations in elderly woman caused by a fall.

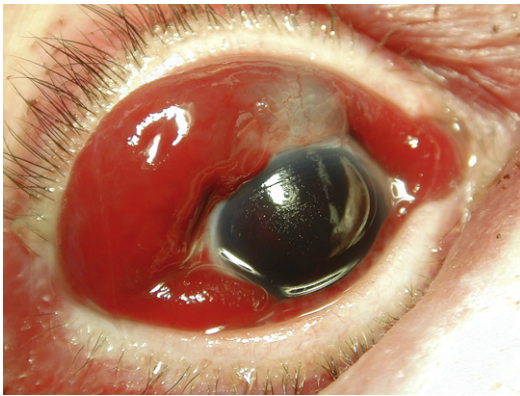


Fig. 16 Right eye of patient in Fig. 12. Lids retracted by applying pressure only to orbital rims. Note pronounced subconjunctival hemorrhage and complete "8-ball" hyphema. Vision was "light perception."



Fig. 17 Blunt injury with lid ecchymosis, and small layered hyphema on the medial aspect of the anterior chamber.

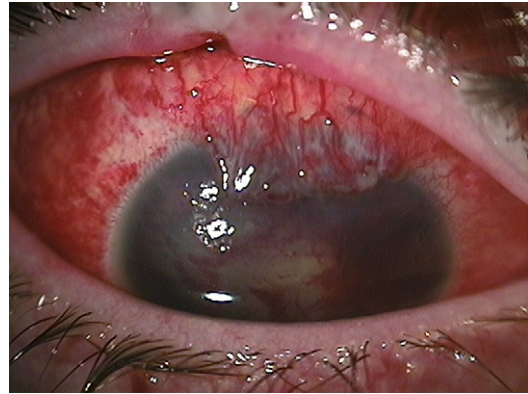


Fig. 18 Severe injury with lens material mixed with blood filling the anterior chamber. Superior lid notch suggests concurrent injury with sharp projectile or small object. This photograph is after a primary surgical repair with conjunctiva sutured over rupture site.

scleral ruptures often have visible intraocular contents that prolapse through the wound. Documenting acuity may seem absurd in such cases but critical and required data. These large ruptures may also have visible anatomic derangement or loss of tissue. The corneal may no longer look round. The conjunctiva has so much hemorrhage under it that the lids do not close. The requirement for emergent consultation is not in question. However, in these cases the presence of a retrobulbar hemorrhage needs to be considered. A tense orbit may only make expulsion of intraocular contents through an open wound worse. A timely and carefully performed lateral canthotomy and cantholysis may preserve an opportunity for vision. Retrobulbar hemorrhages are not synonymous with a ruptured globe but facial trauma severe enough to cause a retrobulbar hemorrhage is severe enough to rupture an eye. Assume the eye is ruptured and avoid any pressure on the eye itself during the examination or an emergent canthotomy.

Treatment awaiting definitive care

After the eye has been examined and the appropriate information obtained, what should one do? The answer is easy. The first step is to protect the eye from any more damage. This is accomplished by placing a plastic or aluminum eye shield over the bony orbit and anchoring it in place with a piece of tape from the forehead to the cheek. For the severely injured eye minimize the patient's activity; when one eye moves, so does the other. Restrict unnecessary physical activities and reading. Consult the ophthalmologist for definitive care arrangements. Do not feed the patient or allow them to drink liquids. This only delays necessary surgery if required. Any ophthalmic medications administered should be at the direction of the ophthalmologist. If the injury is minor enough that urgent or emergent consultation with the ophthalmologist is not required, protection with a shield may not be required. Telephone consultation with the ophthalmologist is reasonable for any injury whose severity is in question. It is much more productive and better for the patient if the referring physician has collected the appropriate data to guide the consultant in an informed decision.

Triage and Management of Cranial Injuries

Meryl A. Severson III, MD^{*}, Randy S. Bell, MD, Rocco A. Armonda, MD

KEYWORDS

• Head injury • Intracranial pressure • Cerebral autoregulation • Cerebral decompression

KEY POINTS

- The optimal evaluation and treatment of the head-injured patient is predicated on initially following established Advanced Trauma Life Support principles and preventing secondary injury.
- Once specific injuries have been identified, maintenance of cerebral perfusion and oxygenation are the keys to maximizing patient outcomes.
- When significant mass lesions are identified or intracranial pressure elevations become refractory to medical intervention, surgical intervention is necessary.

Introduction

Rapid identification, classification, evaluation, and treatment of the head-injured patient is crucial to avoiding secondary injury and potentiation of the initial neurologic insult.¹ This article discusses basic cranial anatomy, classification of head injury, triage decision making, and management concepts for the head-injured patient.

Cerebral anatomy

The skull is composed of more than 25 identified bones however, the cranial compartment is formed primarily by the frontal, parietal, temporal, occipital, and greater wings of the sphenoid bones. The cranial vault is an enclosed space best conceptualized as a rigid, nonexpandable box, subdivided into separate intracranial spaces partitioned by bone as well as dense connective tissue planes. The tentorium cerebelli is a fibrous connective tissue band that separates the intracranial space into supratentorial and infratentorial compartments; the supratentorial space houses the cerebral hemispheres while the infratentorial space contains the cerebellum. The brainstem passes through the tentorial incisura inside the cranial compartment (Fig. 1).

The intracranial space is further divided into the anterior, middle, and posterior fossae, which contain the frontal lobes, temporal lobes, and cerebellum with brainstem, respectively. The right and left supratentorial space is separated by the falx cerebri, nearly identical in composition to the tentorium cerebelli. The brain is divided into the frontal, parietal, occipital, and temporal lobes as well as the midbrain, pons, medulla, and cerebellum (Fig. 2).

Within the brain are ventricles filled with cerebrospinal fluid (CSF), which is produced by the choroid plexus within the ventricular system at a rate of approximately 0.3 mL/min (18–20 mL/h). CSF circulates from the lateral ventricles, to

the foramen of Monro, the third ventricle, the aqueduct of Sylvius, the fourth ventricle, and then out the foramen of Magendie or Luschka. CSF aids in central nervous system (CNS) homeostasis, allows the brain to “float” in the intracranial cavity, and acts as a liquid suspension system, damping normal arterial pulsations. Pathophysiologic obstruction of flow or resorption will result in increased intracranial pressure and hydrocephalus.

The brain receives its blood supply from a right and left internal carotid artery that penetrate the petrous bone, and join with the basilar artery via the posterior communicating artery at the base of the brain, forming the circle of Willis and its associated branches. The middle cerebral artery provides blood supply to the majority of the lateral cerebral hemispheres as well as the base of the temporal lobes. The anterior cerebral arteries are connected by the anterior communicating artery, and provide blood supply to the medial surfaces of the cerebral hemispheres in addition to a medial strip of brain tissue extending from anterior to posterior (Fig. 3). The posterior cerebral arteries provide blood supply to the occipital lobes and a portion of the posterior temporal lobes. The vertebral arteries merge to form the basilar artery, and provide blood supply to the brainstem and cerebellum.

Superficial venous drainage of the brain occurs via (1) cortical bridging veins to the superior sagittal sinus, (2) the vein of Labbe to the superior petrosal sinus, or (3) the basal vein of Rosenthal along the inferomedial border of the temporal lobe to the vein of Galen. Deep drainage of the brain occurs via the deep internal cerebral veins to the vein of Galen, which then empties into the straight sinus. The straight and superior sagittal sinuses combine to form the confluence of the sinuses located at the level of theinion posteriorly; these drain into the transverse and then sigmoid sinuses before emptying into the internal jugular veins. Avoidance of central access to the internal jugular vein ipsilateral to an injured cerebral hemisphere is important in preventing venous occlusion and outflow restriction.

Cerebral physiology

An important physiologic concept is that of intracranial pressure, which is measured in mm Hg or cm H₂O; normal values are

Division of Neurosurgery, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, MD 20889-5600, USA

^{*} Corresponding author.

E-mail address: Meryl.A.Severson.mil@health.mil

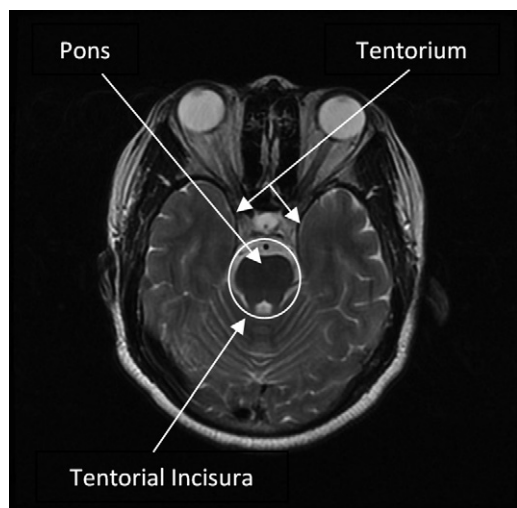


Fig. 1 T2-weighted axial-plane magnetic resonance images demonstrating the brainstem passing through the tentorial incisura. The tentorium is seen as the black bands along the medial border of the temporal lobes. The superior portion of the cerebellum is posterior to the pons.

less than 20 mm Hg. It is important to remember that fluids move from areas of high pressure to low pressure. Intracranial contents include brain tissue, blood (arterial, capillary, venous), and CSF. Addition of a mass lesion to the intracranial space (tumor, hematoma, edema) will cause an increase in intracranial pressure because the skull is rigid and non-expandable. As the mass lesion increases in size, so does the intracranial pressure; as a result CSF is squeezed out of the intracranial compartment via its normal channels which results in reduction of the intracranial pressure (ICP). If the mass continues to expand, venous blood in addition to CSF will be squeezed from the intracranial compartment, keeping ICP within a normal range. If the mass lesion continues to expand, this process will continue to an inflection point that, once passed, sees large increases in ICP with small increases in lesion volume (Fig. 4). As ICP continues to climb, brain tissue will herniate (uncal, subfalcine, or transtonsillar) from its normal anatomic location, causing compression or

displacement of delicate neurologic structures and blood vessels resulting in additional neurologic injury and possibly death. If the pressure is high enough, cerebral perfusion will become impaired because of the pressure gradient required to circulate blood into the intracranial compartment.

The noninjured brain is able to regulate and maintain a near constant blood flow despite changes in blood pressure and vascular volume. This process is termed cerebral autoregulation, which is vital to maintaining adequate cerebral perfusion and, thus, oxygenation. Cerebral perfusion pressure (CPP) is defined as the mean arterial pressure (MAP) minus the ICP. Normal values are approximately 65 to 75 mm Hg.

$$CPP = MAP - ICP$$

The traumatized brain is unable to autoregulate cerebral blood flow, thus hypotension may result in cerebral ischemia whereas hypertension will result in perfusion increases that may cause hemorrhage, edema, or both (Fig. 5). For the intubated head-injured patient ICP monitors are placed into the tissue parenchyma or a drain (termed ventriculostomy) is placed into the ventricular system. These monitors allow second-to-second monitoring of ICP and, thus, CPP when measuring the MAP. Medical therapy can be instituted with a goal CPP target of 65 to 75 mm Hg in the head-injured patient. Ventriculostomies have the added advantage of allowing CSF diversion, and thus direct lowering of ICP in addition to monitoring of intracranial pressure.

Classification of head injuries

Head injuries can be categorized in many ways, including: (1) neurologic status as measured by the Glasgow Coma Score (GCS); (2) whether the injury is open or closed; and (3) whether the injury is a result of a high-velocity or low-velocity impact. Penetrating head injuries may result from low-velocity or high-velocity projectiles and course through a lobe(s) of the brain, across the ventricles (transventricular), or across the supratentorial and infratentorial compartments (bicompartamental) (Box 1).

Head injury is most commonly described in terms of mild, moderate, or severe based on the patient's GCS. It may also be described in terms of open versus closed injuries and further

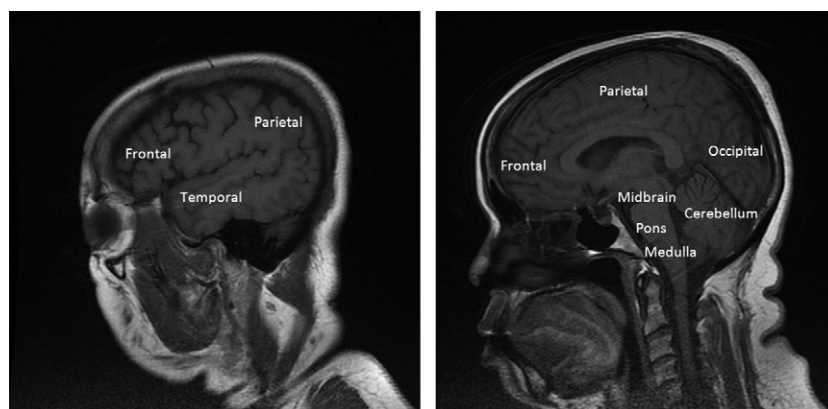


Fig. 2 T1-weighted sagittal images demonstrating the major intracranial central nervous system tissue divisions. The brainstem, composed of the midbrain, pons, and medulla, is located in the posterior fossa with the cerebellum; this space is also referred to as the infratentorial space. The supratentorial space houses the lobes of the cerebral hemispheres and is divided right from left by the falx cerebri. The anterior fossa contains the frontal lobes and the middle fossa contains the temporal lobes; these 2 fossae are separated by the sphenoid ridge.

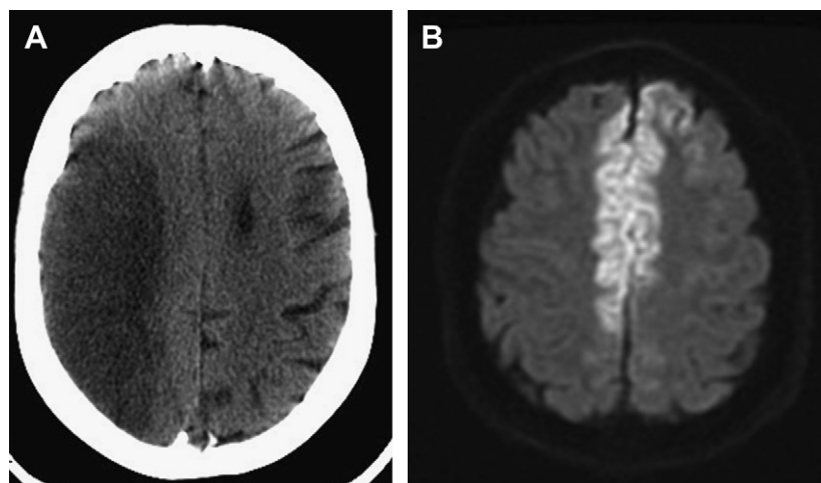


Fig. 3 (A) Noncontrast computed tomography (CT) scan demonstrating infarction of the vascular territory of the right middle cerebral artery. Note the relatively unaffected cerebral tissue supplied by the anterior cerebral artery frontally and in the midline. (B) Diffusion-weighted image of an acute infarction of the anterior cerebral artery bilaterally. These images show the different areas of brain supplied by the anterior and middle cerebral arteries and the lack of collateral supply between major vascular divisions.

subdivided from there. GCS scores have high interrater and intrarater reliability and can be used for prognostication.

Specific patterns of injury may be found commonly between separate categories of head injury. The most common head-injury patterns and their radiologic presentation are discussed here.

Skull fractures are frequently encountered in the head-injured patient. It is imperative to review all studies obtained during imaging, including the bone windows from computed tomography (CT) scans. Fractures of the skull base may present with raccoon eyes, Battle's sign, and rhinorrhea or otorrhea. Traumatic CSF leaks typically resolve spontaneously and may require brief CSF diversion with a ventriculostomy. Fractures of the skull base are frequently associated with maxillofacial fractures. When the energy transfer is significant, severe fragmentation of the anterior cranial fossa floor is common. Skull-base fractures extending to the anterior clinoid process or foramen lacerum, which houses the internal carotid artery, can

result in dissection and stroke. Fractures that cross the draining dural sinuses are to be identified and thoughtfully considered. Surgical exposure in these areas may lead to catastrophic bleeding and worsening of the initial neurologic injury, or possibly result in death from exsanguination.

Parenchymal injuries involve injury to the brain tissue proper and are visually evident on imaging studies (CT and magnetic resonance imaging). Contusions, a common sequela of head trauma, result from rupture of the cerebral capillaries owing to brain impact against the inner skull as well as the soft brain surfaces rubbing over the rough skull base. Contusions at the site of impact are termed "coup" injuries, whereas "contrecoup" injuries are seen opposite the site of impact. Contrecoup injuries result from low pressure opposite the site of impact caused by sudden brain shift.

Intraparenchymal hemorrhages (IPH) result from active bleeding within the brain substance as a result of trauma. IPH may be punctate or large, and can quickly expand, resulting in

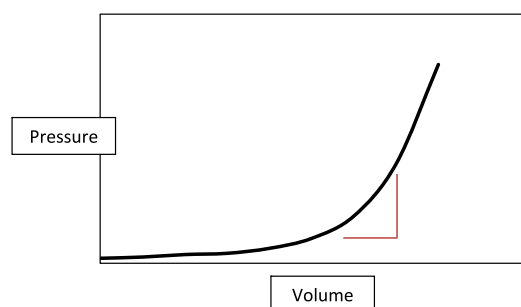


Fig. 4 The inflection point (marked in red) denotes the transition from linear to exponential increases in pressure for increases in volume of a mass lesion (ie, edema, hemorrhage). The intracranial system is no longer able to tolerate further expansion of the mass lesion. Left untreated, the intracranial pressure will quickly increase to catastrophic levels, resulting in further neurologic insult, herniation, and possible death.

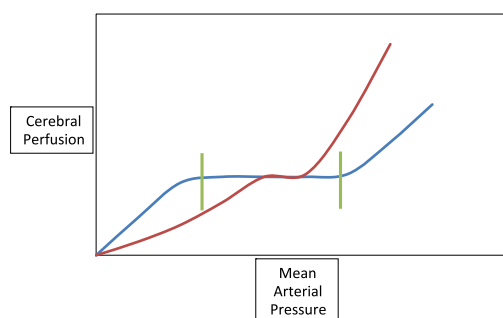


Fig. 5 The noninjured cerebrum (blue line) maintains relatively constant cerebral perfusion across a wide range of blood pressure (green bars). The injured cerebrum is unable to regulate perfusion effectively, resulting in hypoperfusion during hypotension (increased risk of ischemia) and hyperperfusion (increased risk of hemorrhage) with elevated blood pressure (red line).

Box 1. General head injury classifications

Glasgow Coma Score

- 14–15: mild head injury
- 9–13: moderate head injury
- 3–8: severe head injury

Closed Head Injury

- Low velocity
- High velocity

Open Head Injury

- Blunt trauma
 - Low velocity
 - High velocity
- Penetrating trauma
 - Low velocity
 - High velocity
 - Lobar
 - Transventricular
 - Bicompartmental

elevated ICP. Hemorrhages can extend into the ventricular system, and lead to obstructive hydrocephalus and increased ICP. Patients with significant intraventricular hemorrhage (IVH) often require placement of a ventriculostomy for CSF diversion and ICP monitoring.

Violent changes in velocity (velocity is defined as speed and direction of motion) may result in diffuse axonal injury (DAI). The gray matter containing neuronal cell bodies rests on white matter composed of myelinated axons and glia. These tissues have different inertial moments that allow the development of shear forces during trauma, resulting in axonal stretch and rupture. DAI is typically seen at the gray-white junction, corpus callosum, centrum semiovale, and pons, and is treated nonoperatively.

The most common finding after head injury is acute blood in the subarachnoid space, termed traumatic subarachnoid hemorrhage (tSAH), which can be found with relatively minor impacts as well as in severe head injury. The effects are generally self-limited; however, extensive tSAH may result in communicating hydrocephalus, seizure, or even vasospasm of the large to medium-sized cerebral arteries.

Vascular injuries are not as common as brain tissue trauma but can have devastating consequences when present. Arterial dissection results from tearing of the intima and can result in complete or partial vessel occlusion. These injuries are most commonly seen in the internal carotid and vertebral arteries. Complete occlusion commonly results in significant ischemic infarction unless the circle of Willis is intact, allowing perfusion from alternative arterial pathways. Partial occlusions with an intimal “flap” result in thromboembolic events caused by platelet aggregation and intermittent dislodgment. Dissections are common with whiplash injuries and in fractures through the skull base.

Disruption or transection of epidural arteries caused by skull fractures will result in the formation of an epidural hematoma. The dura tightly adheres to the inner table of the skull but can be displaced by systolic arterial pressure and result in an expanding epidural hematoma (EDH). If untreated, the hematoma will often result in compression and displacement of vital

brain tissue, resulting in loss of consciousness and death. The middle meningeal artery is classically described in this situation (Fig. 6).

Venous injuries caused by trauma commonly occur at the bridging veins draining from the cortical surface to the superior sagittal sinus located within the dura at the midline. When these veins are disrupted, blood accumulates in the subdural space, resulting in a subdural hematoma along the cerebral hemisphere. Blood is able to move freely and does not respect the suture lines. Large amounts of blood result in global cerebral hemisphere compression and displacement. Collateral venous channels are not well developed in the CNS. As a result, injury to cortical veins results in impaired venous drainage, causing cerebral venous and capillary congestion. This process can result in ischemia or even hemorrhage, and is termed a venous infarct. As discussed previously, injuries to the dural-based sinuses can be catastrophic if not identified before surgical intervention. When present, these injuries are best treated conservatively.

The scalp is a highly vascular structure that bleeds profusely when lacerated. Short-term control can be obtained with direct pressure or placement of a pressure dressing. Patients without other injuries have progressed to hypovolemic shock from isolated scalp injuries not adequately tamponaded. Definitive control of a profusely bleeding scalp in a traumatic environment can be obtained by placement of full scalp thickness 0-Prolene suture in running fashion.

Cytotoxic edema results from cell injury and death, and is present concurrently with the primary injury. In severe cases edema rapidly progresses, causing severe elevation of ICP. When this occurs it is termed malignant cerebral edema, and necessitates prompt medical therapy and possible surgical intervention in the form of a decompressive craniectomy which is discussed later.

Management principles of the head-injured patient

The appropriate management of the head-injured patient is predicated on avoiding secondary insults that potentiate the primary injury. Evidence has repeatedly shown that outcomes of traumatic brain injury are improved by maintaining oxygen saturations above 90 mm Hg and keeping systolic blood pressure higher than 90 mm Hg at all times with volume resuscitation.¹ Rigorous attention to these 2 vital signs is paramount during the trauma resuscitation of a head-injured patient.

Neuroprotective measures are often instituted to reduce ICP and minimize additional brain injury. These measures include: raising the head of bed to 30° to improve venous return; maintaining euthermia to slight hypothermia to prevent increased cerebral oxygen demand; resuscitating with eutonic or hypertonic fluids to reduce cellular edema and increase vascular volume; maintaining serum sodium levels between 145 and 155 mEq/L; correcting partial thromboplastin time to normal and international normalized ratio to ≤ 1.3 to reduce the risk of cerebral hemorrhage; maintaining a platelet count of 100,000/ μ L to reduce the risk of hemorrhage; initiation of antiepileptic therapy to reduce the short-term seizure risk (the most commonly used medication at this time is levetiracetam however, fosphenytoin and phenytoin are still frequently used); placement of an ICP monitor or ventriculostomy in patients with GCS of 8 or less to allow directed

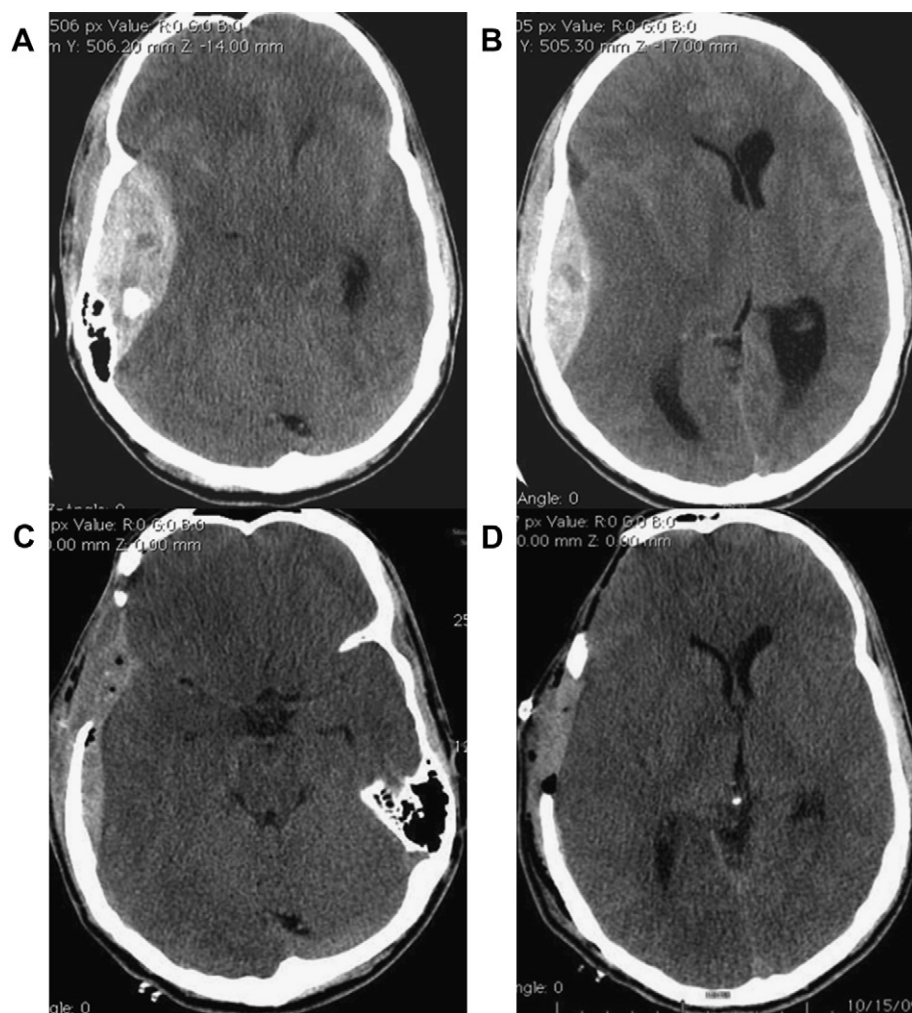


Fig. 6 (A, B) Head computed tomography scan (HCT) showing acute right temporal epidural hematoma caused by disruption of the middle meningeal artery from a skull fracture. The patient underwent emergent hematoma evacuation and craniectomy, resolving the brainstem compression and improving the midline shift (C, D).

medical therapy for ICP reduction and maintenance of normal CPP; and maintaining serum glucose between 110 and 180 mg/dL.

Questions frequently arise regarding the use of steroids in head injury. The Head Injury Guidelines published by the Brain Trauma Foundation review head-injury management recommendations based on levels of evidence. The only recommendation with level I evidence is to avoid the use of steroids in head injury.¹

Specific medical therapy for elevated ICP in the past included fluid restriction and aggressive use of osmotic diuretics such as mannitol. Over the past several years the use of hypertonic saline,² either as a continuous infusion or as a bolus, has increased dramatically as ICP has been shown to vary inversely with serum sodium levels. Hypertonic saline has been used to achieve a specific serum sodium value in some centers and to achieve a target ICP in others. NaCl at 3% is typically used for continuous infusions, whereas greater concentrations, up to 23.5%, are used as bolus therapy. In addition, propofol and dexmedetomidine infusions are used to reduce patient agitation while allowing intermittent neurologic assessments. These agents have rapid on-off sedative effects which is a significant advantage over benzodiazepines.

Evaluation and management of the head-injured patient

Rapid evaluation and identification of the head-injured patient is crucial to understanding a patient's current neurologic status as well as establishing a baseline for successive neurologic comparison. The presence of alcohol, drugs, or other toxins may obscure the initial neurologic examination necessitating more frequent reassessments to monitor neurologic change.

The initial examination and treatment should follow Advanced Trauma Life Support protocol. The neurologic examination should establish the patient's GCS (Table 1).

A GCS of 8 or less (severe head injury) requires emergent intubation and should be performed without pharmacologic muscle relaxants if practicable. Unintended overhyperventilation should be avoided, as reducing the CO₂ extensively has been shown to reduce cerebral perfusion in the head-injured patient. The pupillary examination should be performed with particular attention to size, shape, symmetry, and response to light. Enlarged pupils may indicate a mass lesion with compression of cranial nerve (CN) III against the brainstem, whereas pinpoint pupils may indicate a pontine

Table 1 Glasgow Coma Score

Points	Eye	Verbal	Motor
1	No response	No response	No response
2	Opens to pain	Incomprehensible	Decerebrate
3	Opens to voice	Inappropriate words	Decorticate
4	Open spontaneously	Confused	Withdraws to noxious stimulus
5		Oriented	Localizes noxious stimulus
6			Follows commands

Data from Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet* 1974;2:81–4.

injury. Use of the quantitative pupillometer, a device that precisely and accurately measures the pupil response to a light stimulus, has reduced the subjectivity of pupil evaluation and has been shown to be a reliable predictor of early ICP elevation.³ Visual and manual examination of the head should evaluate for exposed gray matter, scalp lacerations, and/or mobile skull fragments.

During the secondary survey a careful neurologic examination is required. It is important to note whether the patient has received pharmacologic relaxants or depressants as these substances often impede the neurologic examination. The GCS should be repeated and compared with earlier measures. In the awake patient, each CN should be methodically tested and documented. In the obtunded patient, CN reflexes should be examined: pupillary reflex: CN II afferent, CN III efferent; corneal reflex: CN V afferent, CN VII efferent; cough/gag reflex: CN IX afferent, CN X efferent. In cases where brain death is a consideration, the oculoccephalic reflex as well as cold-caloric responses should be assessed. Any dressings or bandages to the head should be removed at this time and the injuries examined. Motor examination of the extremities should be performed identifying specific individual muscle strength if possible. In the head-injured patient this is often not possible, so that only the GCS motor examination can be completed. The sensory examination including deep tendon reflexes should likewise be completed. Upper motor neuron findings often point to severe cranial injuries and should be documented carefully.

Radiologic examination of the patient should be completed as quickly and efficiently as possible. The radiologic test of choice for traumatic head injury is the noncontrast head CT (HCT) followed immediately by a CT angiogram from the aortic arch through the circle of Willis. The HCT should be windowed at W: 80 and L: 40 for optimal evaluation for acute blood, which is bright white on CT. Hemorrhages and contusions are thus hyperdense on CT, whereas ischemic infarcts and edema are hypodense. The CT can be windowed to W: 3500 and L: 500 for evaluation of bony fractures. The CT angiogram should be critically examined in the axial plane specifically identifying the origin, course, and termination of the cervical and intracranial vessels. Vessel cutoffs, narrowings, or intimal flaps should be noted and examined on the reconstructed sagittal and coronal images. Cervical fractures increase the likelihood of extracranial arterial dissections, whereas skull-base fractures are frequently associated with carotid dissection and injury.

The management steps for a patient with a GCS of 8 or less is to establish definitive airway control via endotracheal intubation. A neurologic examination is completed and an HCT obtained. Patients without a mass lesion should undergo

Table 2 Comparison of mortality percentages in patients with moderate to severe head injury according to HCT findings when classified using the Marshall Classification in comparison with the Rotterdam CT score

Marshall Classification	Mortality (%)
I. Diffuse injury I	6.4
a. No cerebral pathology on CT	
II. Diffuse injury II	11
a. Lesions identified on CT and/or (b)	
i. No high or mixed density lesions >25 cm ³	
ii. May include bone fragments and foreign bodies	
b. Cisterns present with midline shift 0–5 mm	
III. Diffuse injury III	29
a. Cisterns compressed or absent	
b. Midline shift 0–5 mm	
c. No high or mixed density lesion >25 cm ³	
IV. Diffuse injury IV	44
a. Midline shift >5 mm	
b. No high or mixed density lesion >25 cm ³	
V. Evacuated mass lesion	30
VI. Nonevacuated mass lesion	34
a. High or mixed density lesion >25 cm ³	

Rotterdam CT Score	Points	Score	Mortality (%)
Basal Cisterns		1	0
Normal	0	2	6.8
Compressed	1	3	16
Absent	2	4	26
		5	53
		6	61
Midline Shift			
0–5 mm	0		
>5 mm	1		
Epidural Mass Lesion			
Present	0		
Absent	1		
Intraventricular Blood or Traumatic Subarachnoid Hemorrhage			
Absent	0		
Present	1		
Sum Score	+1		

The Rotterdam CT Score is increasingly being used for general prognostication in head injury.

Data from Maas AI, Hukkelhoven CW, Marshall LF, et al. Prediction of outcome in traumatic brain injury with computed tomographic characteristics: a comparison between the computed tomographic classification and combinations of computed tomographic predictors. *Neurosurgery* 2005;57(6):1173–82.

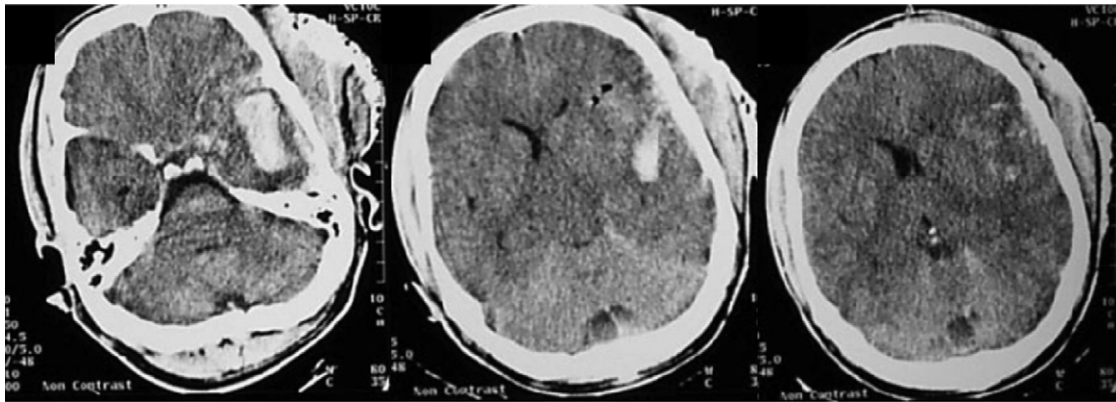


Fig. 7 Emergent HCT of patient in Fig. 8 showing left temporal hematoma, loss of basal cisterns, compression of the left cerebral peduncle, edema of left cerebral hemisphere, and left-to-right midline shift.

emergent placement of a ventriculostomy or ICP monitoring device. Nonsurgical ICP management strategies (discussed earlier) are then implemented. Patients with an open head injury, depressed skull fracture, or significant mass lesion should be taken to the operating room for surgical treatment of their primary injury as well as placement of either a ventriculostomy or ICP monitoring device. Postoperatively the neuroprotective measures detailed previously should be instituted.

When ICP becomes elevated and does not improve with medical management, a repeat emergent HCT without contrast to evaluate for new or worsening cerebral findings is mandatory. If the HCT is unchanged, the patient should be evaluated with an electroencephalogram (EEG) for nonconvulsive status epilepticus⁴ even if the patient is already receiving antiepileptic medications (AEDs). If seizure activity is identified, it should be broken quickly with intravenous lorazepam and the current AED dosing increased or, if already at maximum dosing, a second agent added.

Alternatively, if the HCT shows an expanding mass lesion with shift, prompt evacuation of the mass lesion should be performed. If the suspicion for worsening cerebral edema over the next several days is high, consideration should be given to performing a generous craniectomy (ie, skull removal) at the same time for maximal cerebral decompression. In instances of temporal lobe trauma, removal of the anterior 3 cm of the temporal lobe is often performed to maximally decompress the middle fossa and prevent possible brainstem compression.

When the emergent follow-up HCT shows worsening contusions and/or edema, consideration for craniectomy and cerebral decompression should be given. Cerebral decompression may be achieved by performing a large 15 × 12-cm craniectomy of the frontal, parietal, and temporal bones, termed a hemicraniectomy (see Case 1). A bifrontal craniectomy involves removal of the frontal bones and is used in instances of bifrontal lobe injury (see Case 2). In conjunction with a craniectomy, the dura is opened, allowing further decompression of the cerebral tissue, and covered with a dural substitute. An alternative treatment option is to institute barbiturate coma. The patient is loaded and placed on a continuous infusion of a barbiturate medication such as pentobarbital. The patient is simultaneously connected to continuous EEG monitoring with the goal of achieving a 10- to

15-second “burst suppression” of cerebral activity; this means the EEG tracing is flat for 10 to 15 seconds followed by a very brief burst of neuronal activity. By suppressing neuronal activity, the metabolic demands of the cerebral tissue are decreased, thereby reducing neuronal injury or death due to elevated ICP. The ICP and drug level are followed closely to monitor the effectiveness of therapy. Barbiturates have long half-lives and continue to have CNS-depressant effects for several days following cessation. Medically induced coma may also be instituted following cranial decompression in patients with persistently elevated ICP.

When proceeding to the operating room with a head-injured patient, whether emergently or electively, it is critical for the anesthesia and surgical teams to remain hyperaware of the patient’s CPP. Initiating general anesthesia can cause systemic hypotension, leading to disastrous neurological consequences in a patient with elevated ICP and impaired cerebral autoregulation. Often the patient will require vasoactive medications to support systemic pressure and, thus, CPP while under general anesthesia. The surgical team must communicate with anesthesia frequently with regard to blood loss as well as



Fig. 8 Intraoperative photograph of a large left-sided soft-tissue defect following blast trauma. This patient presented in neurologic extremis and required emergent neurosurgical intervention.

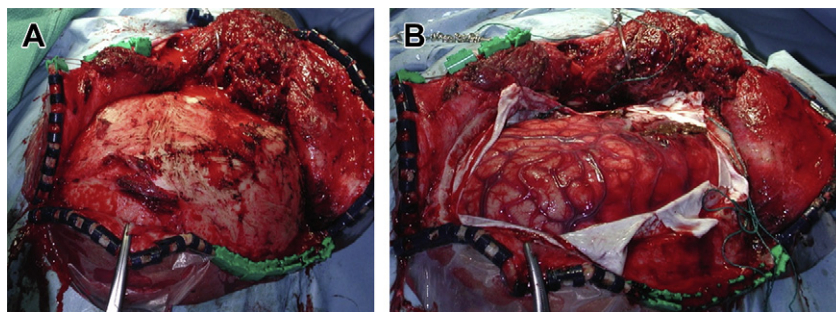


Fig. 9 Intraoperative photographs of the patient in Fig. 8 showing cranial exposure after incision (A) and left cerebral hemisphere exposure following hemicraniectomy and stellate dural opening, allowing maximal decompression (B). The patient's head is turned to the right and is in a lateral position. Superior is toward the bottom of the photographs and anterior is toward the right borders.

timing of bone removal and dural opening, as cardiovascular collapse can occur with these maneuvers.

CT findings in head trauma have been correlated with mortality. The Marshall Classification and, more recently, the Rotterdam CT score are 2 measures that have been used in this capacity.⁵ The Rotterdam classification is increasingly being used for general prognostication in head injury and emphasizes the better prognosis associated with a promptly evacuated EDH compared with a parenchymal injury (Table 2).

Surgical management of selected head injuries

The indications for neurosurgical intervention depend on the nature of the head injury and the patient's neurologic status. Advanced Trauma Life Support (ATLS) resuscitation protocols should be followed rigorously. Patients with severe head injury (GCS ≤ 8) require emergent intubation and HCT as soon as practicable. Those exhibiting clinical signs of herniation (Cushing reflex [hypertension, bradycardia, irregular respirations], unilateral dilated unreactive pupil, motor posturing) should be simultaneously treated with maximal medical therapy to reduce potentially elevated ICP. Significant mass lesions (EDH, subdural hematoma, intraparenchymal hematoma

[IPH]), no matter the mechanism associated with neurologic compromise and/or elevated ICP require prompt surgical evacuation with consideration for craniectomy in the trauma setting.

Case 1. Lateral cranial blast injury

This patient suffered a blast injury and was found to be unresponsive with a GCS of 3. He was emergently intubated and resuscitated in accordance with Combat Casualty Care protocols and rapidly evacuated to a field hospital with neurosurgical capability. His neurologic examination on arrival revealed: a degloving injury to the left face; a dilated and nonreactive left pupil; intact right corneal reflex; intact cough reflex with deep suctioning; and decorticate posturing bilaterally. He was briefly hyperventilated and started on a 3% saline infusion at 50 mL/h.

Emergent HCT showed a left temporal IPH with effacement of the basal cisterns, compression of the left cerebral peduncle (uncal herniation), edema of left cerebral hemisphere, left-to-right midline shift, and significant craniofacio-orbital bony and soft-tissue injury of the lateral orbit, and temporal and middle cranial fossae (Fig. 7).

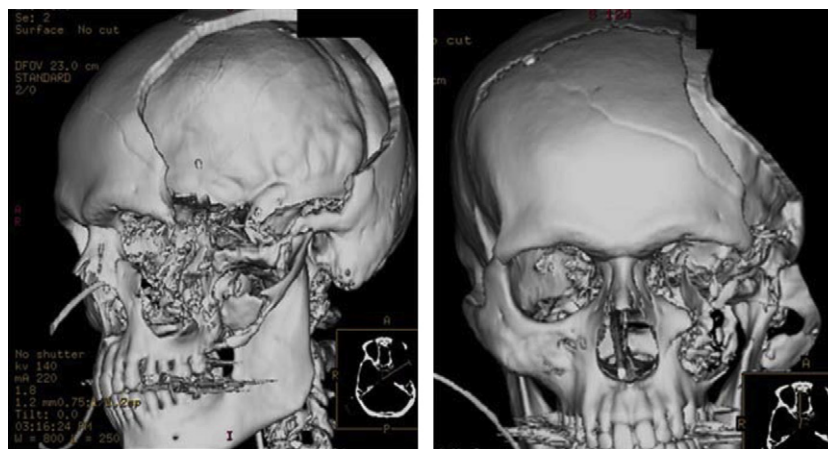


Fig. 10 Three-dimensional bony CT reconstruction of the patient in Fig. 8 following his operative procedure. Note the severe bony disruption of the lateral orbital wall, maxilla, and temporal fossa. These bony defects require extensive reconstruction and are best managed in a delayed fashion.

The patient was taken emergently to the operating room (Fig. 8) because of his poor neurologic examination consistent with early herniation and findings on HCT. The surgical objectives acutely were to (1) remove the IPH from the left temporal lobe thereby relieving the brainstem compression (herniation); (2) perform a large decompressive craniectomy to allow for cerebral swelling over the next several days; and (3) place an external ventricular drain to allow for ICP management with CSF diversion. Definitive correction of his craniofacial bony defects was performed in a delayed fashion.

His loss of craniofacial tissue posed a significant challenge to the necessary cranial decompression, soft-tissue anatomic continuity reconstruction, and ability to obtain a normal delayed contour. A large curvilinear incision was made outside the zone of injury from the posterior aspect of the soft-tissue defect to the midline anterior hairline (Fig. 9). This incision allowed wide exposure of the left hemi-skull, completion of a large hemicraniectomy (Fig. 10), removal of the temporal IPH, and repair of the skull base with packing and delayed closure of the infratemporal fossa. The patient's hospital course was prolonged and complicated. However, he ultimately survived his injuries and is currently living independently.

Case 2. Anterior cranial blast injury

This patient was wounded in a frontal mortar blast. He was resuscitated in the field in accordance with Combat Casualty Care protocols and rapidly evacuated to a field hospital with neurosurgical capability. On arrival his GCS was 8 (E1, V2, M5); his left pupil was 3 mm and reactive with an intact corneal reflex; his cough reflex was intact. Clearly seen was a severe craniofacio-orbital blast injury with traumatic right eye enucleation (Fig. 11).

CT imaging was obtained and showed bifrontal parenchymal contusions with edema, tSAH, indriven bony fragments, multiple craniofacial fractures including loss of the anatomic continuity of the anterior skull base, orbital bandeau, disruption of the frontal sinuses, and a Le Fort III fracture (Fig. 12).

He was taken emergently to the operating room with the following objectives: (1) irrigate, debride, and close the

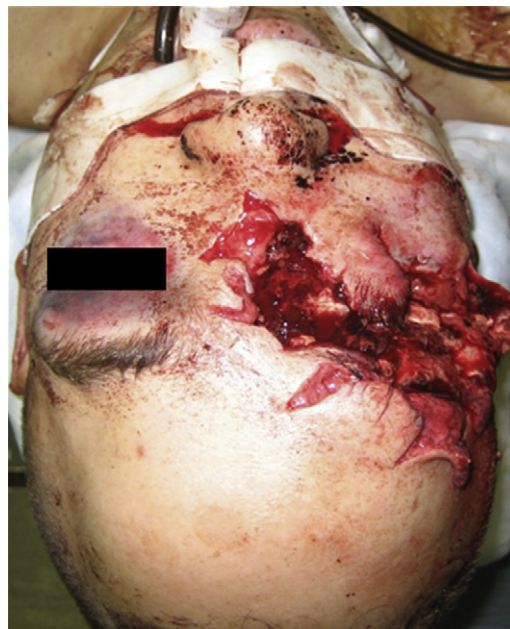


Fig. 11 Severe craniofacio-orbital trauma resulting from a mortar blast to the right face.

primary injury site; (2) perform a bifrontal decompression to allow cerebral swelling; (3) preliminary orbital bandeau reconstruction to maintain basic craniofacial structure in anticipation of future reconstruction; (4) expedited anterior cranial floor reconstruction to restore anatomic continuity and cerebral support; and (5) placement of an external ventricular drain for ICP measurement and CSF diversion. His Le Fort III fracture was addressed in delayed fashion (Figs. 13 and 14).

Two weeks after injury the patient underwent diagnostic cerebral angiography (Fig. 15) to evaluate for occult vascular injury, which is frequently seen in patients with cerebral blast and penetrating injuries. A pseudoaneurysm was identified on a major arterial branch from the right callosomarginal artery,

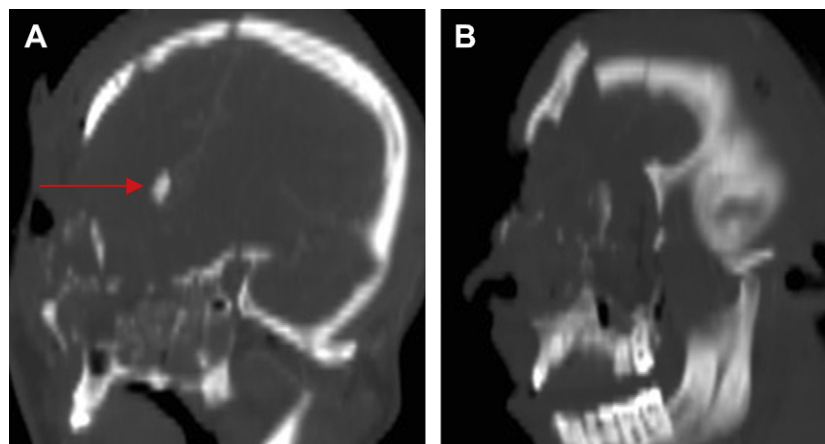


Fig. 12 Selected coronal (A) and sagittal (B) reconstruction images obtained from initial postinjury CT. Note the severe disruption of the right anterior fossa floor, orbital bandeau, and frontal sinus. The red arrow depicts an indriven bony fragment adjacent to arterial tributaries from the right callosomarginal artery.

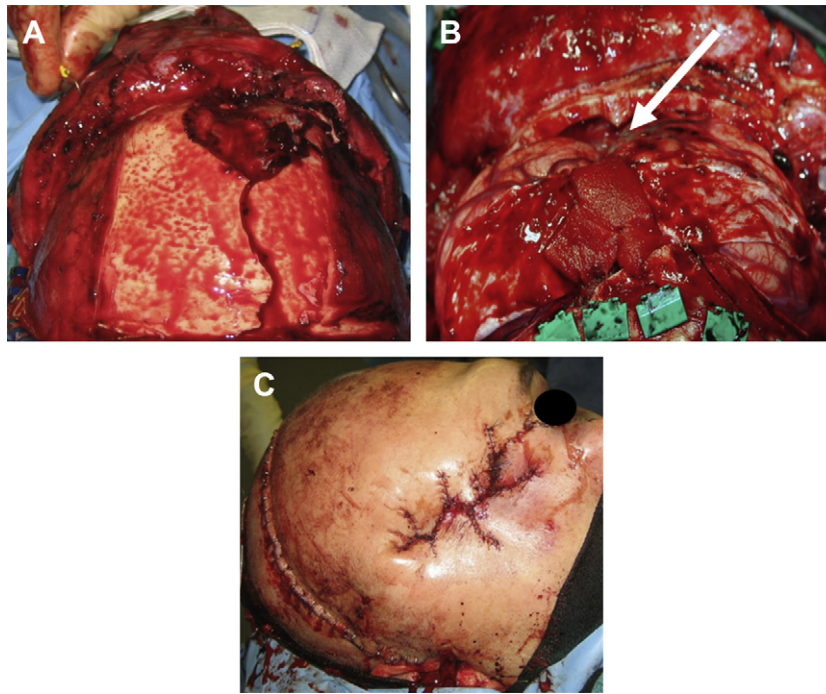


Fig. 13 (A) Intraoperative photograph of the patient in Fig. 11 with bicoronal incision and associated large "tectonic-plate" fractures of the frontal bone and orbital bandeau across the frontal sinuses. (B) Intraoperative photograph of a different patient undergoing a bifrontal craniectomy; note the protrusion of the frontal lobes as the dura is incised owing to diffuse cerebral edema. The floor of the anterior cranial fossa is also visualized (arrow). (C) Intraoperative photograph of the patient in Fig. 11 at the completion of a bifrontal damage control decompressive craniectomy, preliminary orbital bandeau repair, anterior fossa floor reconstruction, right eye enucleation, placement of external ventricular drain, and closure.

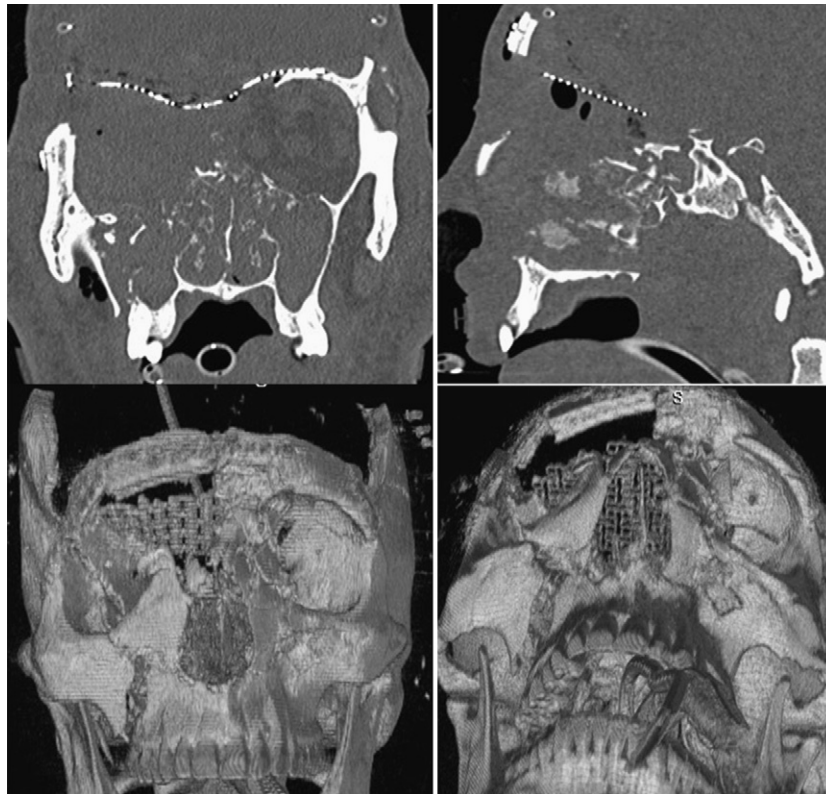


Fig. 14 Postoperative CT images and 3-dimensional reconstructions of the patient in Fig. 11. The anterior fossa floor was reconstructed with titanium mesh and the right orbital bandeau was preliminarily reconstructed using autologous cranial bone graft. The patient's Le Fort III fracture was repaired in delayed fashion. Previously it was the authors' preference to use titanium mesh, as was done in this case. Delayed complications with mesh have occurred and reoperation has been challenging. The authors now use split-thickness calvarial graft instead of mesh, when and where possible, when reconstructing the cranial floor and orbital bandeau.

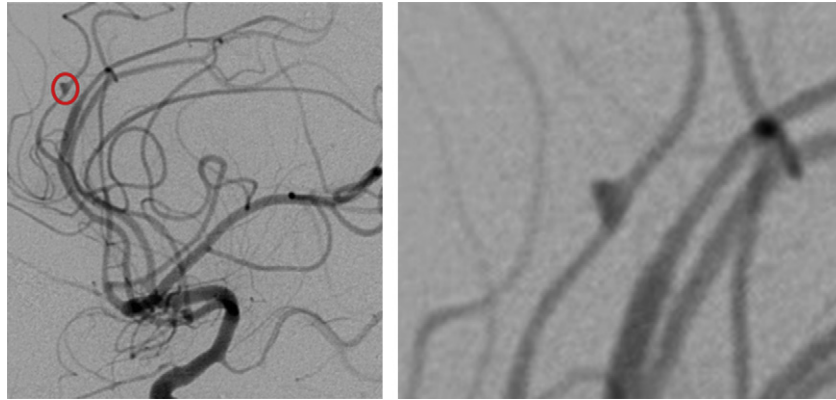


Fig. 15 Lateral view of a mid-arterial phase right internal carotid artery angiogram showing a vessel abnormality of an arterial branch from the right callosomarginal artery (*red circle*). This anomaly occurred at the location of the indriven bone fragment identified in [Fig. 12A](#). The pseudoaneurysm was treated with placement of endovascular coils.

and was treated. The patient survived his injuries and is currently living independently.

Summary

This article discusses basic head and intracranial CNS anatomy, cerebral physiology, and classifications of head injury. Management principles and the practice of head-injury evaluation are reviewed and supplemented by the presentation of selected head-injury scenarios. The optimal evaluation and treatment of the head-injured patient is predicated on initially following established ATLS principles and preventing secondary injury. Once specific injuries have been identified, maintenance of cerebral perfusion and oxygenation is the key to maximizing patient outcomes, and this often requires measurement and treatment of ICP values. When significant mass lesions are identified or ICP elevations become refractory to medical intervention, surgical intervention is necessary.

References

1. A joint project of the Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons, AANS/CNS Joint Section on Neurotrauma and Critical Care. Bullock MR, Povlishock JT, editors. Guidelines for the management of severe traumatic brain injury. *J Neurotrauma* 2007;24(Suppl 1):S1–106.
2. Mortazavi MM, Romeo AK, Deep A, et al. Hypertonic saline for treating raised intracranial pressure: literature review with meta-analysis. *J Neurosurg* 2012;116:210–21.
3. Chen JW, Gombart ZJ, Rogers S, et al. Pupillary reactivity as an early indicator of increased intracranial pressure: the introduction of the Neurological Pupil index. *Surg Neurol Int* 2011;2:82–8.
4. Brophy GM, Bell R, Alldredge B, et al. Guidelines for the evaluation and management of status epilepticus. *Neurocrit Care* 2012;17:3–23.
5. Maas AI, Hukkelhoven CW, Marshall LF, et al. Prediction of outcome in traumatic brain injury with computed tomographic characteristics: a comparison between the computed tomographic classification and combinations of computed tomographic predictors. *Neurosurgery* 2005;57:1173–82.

Otologic and Temporal Bone Injuries, Triage, and Management

Isaac D. Erbele, MD*, M. Peter Sorensen, MD , Arnaldo Rivera, MD

KEYWORDS

• Temporal bone • Trauma • Facial nerve

KEY POINTS

- Temporal bone trauma requires a large amount of force, meaning that patients will presents with multiple injuries and the temporal bone injury is often incidentally found. Focus should first be on assessing and treating the ABCs before management of temporal bone injuries.
- The physical examination should include inspection of the face and soft tissue for lacerations, assessment of cerebrospinal fluid leakage, assessment of the facial nerve, an otoscopic examination of the ear, a tuning fork examination with a 512-Hz tuning fork, and a complete cranial nerve examination.
- If a temporal bone fracture is suspected, a fine-cut computed tomography scan of the temporal bone is indicated.
- One of the most important physical examination findings for patients with temporal bone injuries is function of the facial nerve immediately after the injury. This must be assessed as soon as it is possible, and an assessment may be performed in an unconscious patient.
- If the facial nerve is not functioning immediately following the injury, workup by an otolaryngologist for potential facial nerve exploration is indicated.
- If a cerebrospinal fluid leak is identified from the ear or from the nose, workup by an otolaryngologist and neurosurgeon is indicated.
- Early audiology consultation is indicated if there is any hearing loss.
- Conductive hearing loss immediately following these injuries can be a result of traumatic tympanic membrane perforations, hemotympanum, cerebrospinal fluid in the middle ear, or ossicular chain disruption.
- Traumatic sensorineural hearing loss is typically the result of noise-induced trauma, otic capsule disruption, or perilymphatic fistulas.

Introduction

Injury to the temporal bone requires a great deal of force: the lateral force required for a temporal bone fracture is estimated at greater than 1875 lb of force.¹ Trauma may result in fractures through the temporal bone, including through the otic capsule, injury to the facial nerve, ossicular chain discontinuity, tympanic membrane perforation, perilymphatic fistulas, cerebrospinal fluid (CSF) leaks, and injuries to the carotid artery.

Temporal bone contents can be injured by blunt trauma, penetrating trauma, and barotrauma. The most common cause of temporal bone trauma in civilian hospitals is motor vehicle accidents, followed by assaults and falls.² Gunshot injuries to the temporal bone are less common. Within the military population, tympanic membrane perforations caused by blast injury are fairly common. A series from Brooke Army Medical

Center found that 16% of patients sustaining blast injuries had tympanic membrane perforations.³

In the case of a gross CSF leak or hemorrhage from the ear, emergent action is necessary. Otherwise, injury to the temporal bone is infrequently life-threatening in and of itself. Given the amount of force required for a temporal bone injury, concomitant injuries are common, and attention should be paid to any airway, breathing, and circulation injuries first.

This article explores the initial evaluation and management of temporal bone trauma and auricular injuries.

History

There are a number of critical pieces of historical information that the facial trauma specialist needs to obtain when evaluating a patient with head, neck, and temporal bone trauma. Often for the isolated temporal bone fracture discovered from a routine trauma head computed tomography (CT) scan, this information can be obtained from the patient, but care should be taken to collect this information from alternative sources if the patient is unconscious or otherwise unable to communicate.

The mechanism of injury and associated injuries should be obtained first. Next, it should be determined if there is or was any facial nerve function after the injury, for reasons will we

The authors have nothing to disclose.

Previous Presentation: None.

Department of Otolaryngology Head and Neck Surgery, Walter Reed National Military Medical Center, Bethesda, MD, USA

* Corresponding author. 8901 Rockville Pike, Bethesda, MD 20889.

E-mail address: Isaac.d.erbele.mil@health.mil

go into further later in this article. To assess injuries to the otic capsule and the cochlear nerve, determine if there is hearing loss, tinnitus, or vertigo. Ask specific questions regarding CSF leaks, including otorrhea, watery rhinorrhea, and salty taste in the back of the mouth.

Physical examination

A facial trauma specialist should perform a complete head and neck examination. Grossly, the head and neck should be evaluated for CSF leaks and otorrhea. The skin should be examined for abrasions, laceration, and open fractures, including evaluating the entire scalp for lacerations. Identify lacerations and hematomas of the auricle. Hematomas, particularly those involving the auricle, should be drained and bolstered to prevent “cauliflower ear.” Postauricular and periorbital ecchymosis should be identified and recorded, as these can be evidence of a basilar skull fracture. Anterior nasal endoscopy with a standard otoscope should be performed to rule out gross CSF rhinorrhea.

A complete examination also includes otoscopy, a tuning fork examination, a facial nerve examination, and comprehensive cranial nerve examination (Fig. 1).

Otoscopy

The otoscopic examination is one of the most important components of the examination for temporal bone trauma, but it can also be the most challenging because of debris in the ear canal, the inability of the patient to be able to turn his or her head because of cervical spine stabilization, and operator inexperience with the otoscopic examination.

The angle of the external auditory canal becomes larger and more horizontal as the patient ages. For newborns and small children, it may be necessary to gently tug the pinna posteroinferiorly with a small (size 3 mm or 4 mm) otoscopic speculum for adequate evaluation. Adults can typically accommodate a larger speculum (size 5 mm), but may still require gentle tugging of the pinna to obtain an adequate view of the tympanic membrane.



Fig. 1 Basic tool kit for initially assessing temporal bone trauma. Note the 512-Hz tuning fork on the left, the otoscope in the center, and the various sizes of specula on the right. The smallest specula (3 mm and 4 mm, on the bottom and middle right, respectively), are typically used in small children and infants.

The external auditory canal should be evaluated for any lacerations, canal edema, debris, blood, or CSF. Some of the debris may only be cerumen (Fig. 2). Cerebrospinal fluid is typically a thin, clear liquid. Careful examination of the external auditory canal may also reveal fractures of the temporal bone visible through the thin skin of the canal wall. This is also important to document. Occasionally, the external auditory canal wall skin can grow into the fracture, resulting in a canal wall cholesteatoma years later.

The tympanic membrane, if visible, should then be assessed (Fig. 3). Fluid behind the tympanic membrane should be identified. Clear fluid, occasionally with air bubbles within the fluid, can suggest CSF, although it may also suggest a serous otitis, particularly in a child. Hemotympanum will be obvious as dark red or black fluid behind the tympanic membrane. The tympanic membrane should also be assessed for perforation. The perforation should be described based on the quadrant and the percentage of the tympanic membrane perforated (Fig. 4).

Tuning fork examination

In temporal bone trauma, hearing loss is a common complaint. Hearing loss can be divided into 2 broad categories that can help narrow the differential diagnosis. Conductive hearing loss occurs because of obstruction of the sound waves from reaching the tympanic membrane, noncompliance or perforation of the ear drum, or poor of movement of the ossicular chain. Sensorineural hearing loss is caused by injuries to the cochlea or to the cochlear nerve.

The tuning fork examination is a simple examination that can rapidly determine the type of hearing loss a patient has. There are 2 parts to this examination, the Weber test and the Rinne test. Both should be used as a piece of information within the clinical context of the patient, and not used in isolation.

These examinations require a conversant patient. For both, a 512-Hz tuning fork is preferred. Care is taken to strike the tuning fork on a soft surface to prevent high-pitched overtones.

In the Weber test, an oscillating tuning fork is placed in the midline, typically on the maxilla or forehead. The most reliable results, however, come when the tuning fork is placed on the upper incisors (Fig. 5). In conductive hearing loss, the patient will hear the sound of the vibrating tuning fork louder on the affected side. In sensorineural hearing loss, the patient will

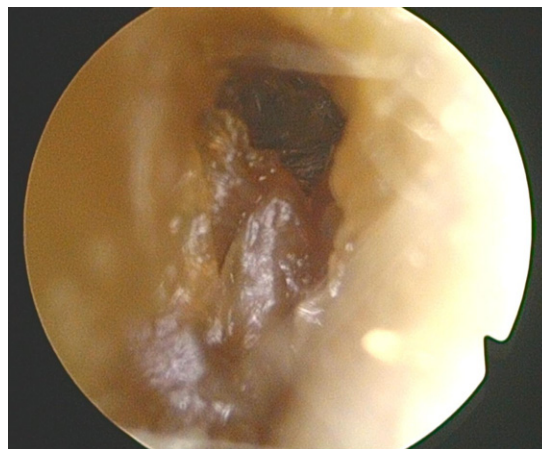


Fig. 2 Cerumen debris on otoscopic examination. Remove under binocular microscopy to allow for appropriate view of the tympanic membrane.

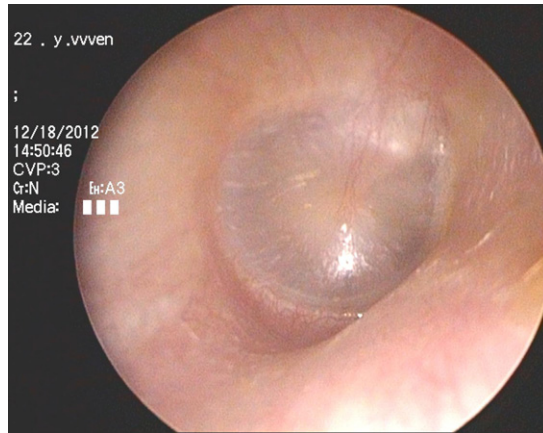


Fig. 3 This is the appearance of a normal, healthy appearing right tympanic membrane. The tympanic membrane is translucent, there is no red or brown discoloration behind it, and there is no air fluid level or air bubbles.

hear the tuning fork louder on the contralateral side. If a patient can hear the sound louder in one ear (typically the difference of 3–5 dB), the Weber is said to lateralize to that side. If the patient hears the sound in the middle, it is said to be midline or normal.

The Rinne test is used in conjunction with the Weber (Fig. 6). The oscillating tuning fork is first placed on the patient's mastoid and then right in front of the ear. The patient is then asked which they hear louder. If they hear the sound louder with the tuning fork on the mastoid, bone conduction (BC) is said to be greater than air conduction (AC). In normal, or positive, Rinne, AC is greater than BC. At our institution, we record this as $AC > BC$, $BC > AC$, or $AC = BC$. The test then is repeated for the contralateral ear. If BC is louder than AC, this suggests a conductive hearing loss of greater than 20 dB. AC greater than BC is found in either a normal-hearing ear or in the presence of sensorineural hearing loss.

For example, if a patient has decreased hearing in the right ear, and the Weber test lateralizes to the left ear with a normal



Fig. 4 This is a view of a patient's right ear with extensive myringosclerosis, evident as a chalky appearing tympanic membrane, as well as having a 40% anterior-inferior tympanic membrane perforation.



Fig. 5 The Weber test is performed with a 512-Hz tuning fork on a midline structure, and it can distinguish a 3-db difference between ears. The most reliable location for this test is on the premaxillary incisors.

Rinne in both ears ($AC > BC$), this would suggest the patient has a sensorineural hearing loss. On the other hand, if the Weber test lateralized to the right side, and the Rinne was negative ($BC > AC$) on the right ear, this would suggest a conductive hearing loss instead.

Facial nerve examination

The first and most important consideration for the facial nerve is to determine timing of a facial nerve injury. Often, immediate loss of facial nerve function suggests transection or severe compression of the nerve. Gradual loss, however, suggests edema or reactivation of a virus. This is information that guides whether or not surgical intervention is indicated. Therefore, it is essential to obtain an adequate facial nerve examination when the patient initially presents, preferably before muscle relaxants are given and the patient is intubated.

The facial nerve's principal responsibility is to control the muscles of facial expression. Loss of function is disfiguring and can cause significant morbidity. The motor branch of the facial nerve has 5 major branches: temporal, zygomatic, buccal, marginal mandibular, and cervical. Asymmetry across branches is worrisome for a facial nerve injury within the temporal bone, but individual extracranial branches may also be injured in facial trauma. Identifying the injured branches helps drive surgical management, as we explore later in this article. Testing is simple in the conscious patient:

1. Asking patients to raise their eyebrows tests the frontalis muscle, which is enervated by the temporal branch.
2. Asking patients to close their eyes tests the orbicularis oculi, which is enervated by the zygomatic branch.
3. Asking patients to smile tests a variety of muscles, but particularly the zygomaticus major and minor, which are enervated by the buccal branch.
4. Asking patients to say "e" or to show their lower teeth tests the depressor anguli oris, which is enervated by the marginal mandibular branch.
5. The cervical branch enervates the platysma. Injury to this branch can be safely ignored (Fig. 7).

If patients are not interactive, a painful stimulus, such as a sternal rub or ungual compression, should be used to assess their grimace. Although not ideal, this is preferred over obtaining an inadequate examination.

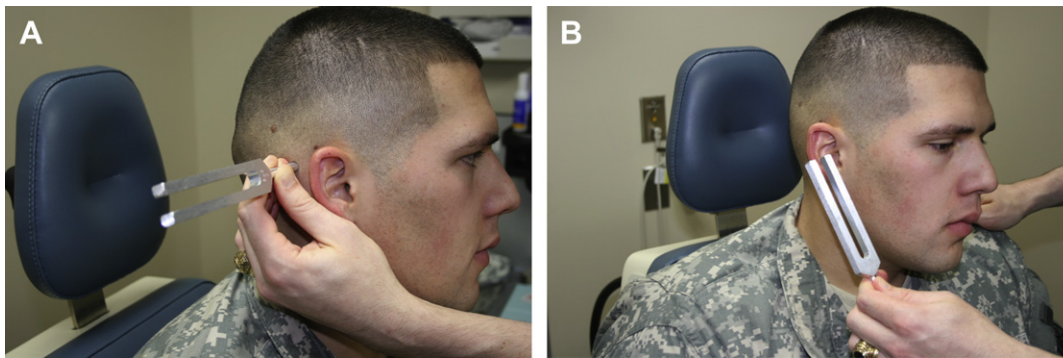


Fig. 6 The Rinne test is performed first with the tuning fork on the mastoid (A), followed by placing it in front of the ear (B). The patient is then asked which is louder. In a patient with conductive hearing loss, placing the tuning fork on the mastoid (A) will appear to sound louder than placing it in front of the ear (B); in other words, bone conduction will be greater than air conduction.

For facial nerve injuries involving all extracranial branches, the House-Brackmann grading system is used. There are limitations to this grading system, not the least of which includes interobserver and intraobserver variability, but it has been adopted as the standard grading system of facial nerve disorders by the American Academy of Otolaryngology.⁴ The grading system ranges from normal (House-Brackmann I) to complete paralysis (House-Brackmann VI) (Table 1).⁵

One of the most important findings clinically is the ability to close the eye. In the House-Brackmann grading system, this is the difference between grade III and grade IV. Additionally, if an injury of the facial nerve is proximal enough, it may affect the

parasympathic innervation of the lacrimal gland as well. A combination of poor lacrimation and failure to completely close the eye can lead to corneal desiccation and potentially blindness.

Lacrimation can be tested with the Schirmer test, but this does not have a role in the acute management of facial nerve injuries. The sublingual and submandibular glands are also innervated by the facial nerve, but unilateral dysfunction of the sublingual and submandibular glands is not typically significant clinically. Taste disruption of the anterior two-thirds of the ipsilateral tongue is also possible with facial nerve injuries, but it also is not typically significant clinically.

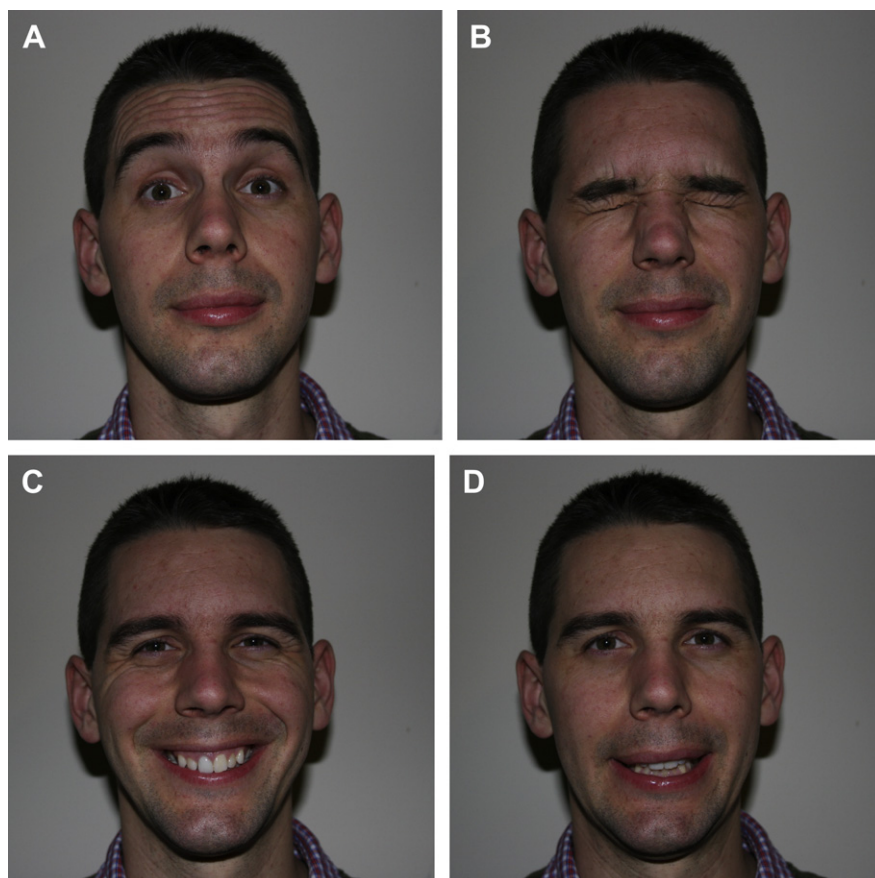


Fig. 7 The facial nerve is tested by examining each of its major branches: the frontal branch (A), the zygomatic branch (B), the buccal branch (C), and the marginal mandibular branch (D). The cervical branch is not routinely tested.

Table 1 House-Brackmann grading system for the degree of nerve damage in facial nerve palsy

Grade	Description	Measurement	Function %	Estimated Function %
I	Normal	8/8	100	100
II	Slight	7/8	76–99	80
III	Moderate	5/8–6/8	51–75	60
IV	Moderately severe	3/8–4/8	26–50	40
V	Severe	1/8–2/8	1–25	20
VI	Total	0/8	0	0

Adapted from House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg 1985;93(2):146–7; with permission.

Vestibular examination

Spontaneous nystagmus in the first several days after the trauma, particularly in the presence of unilateral hearing loss, suggests the presence of a fracture through the vestibular system. The direction of the nystagmus, the fast phase of the eye movement, will be away from the injured vestibular system.

The most common vestibular sequelae of temporal bone trauma, however, is benign paroxysmal positional vertigo, a form of peripheral vertigo that is hypothetically caused by disruption of otoliths into the semicircular canals. This process presents as seconds of rotational vertigo, particularly after sudden movements of the head. This can be assessed with a Dix-Hallpike maneuver once the patient is stable and their cervical spine has been cleared. To perform this examination, the patient's head is turned 45° to the side, and the patient is rapidly brought from a sitting position to lying position, allowing the head to hang below the table toward the tested side. A positive test occurs when, within 30 seconds, the patient has dizziness and nystagmus. The ear to the ground is the effected vestibular system.⁶

Central vertigo, which is attributable for instance to traumatic brain injuries instead of trauma to the vestibular system, may also occur. Central vertigo will present with nystagmus that does not suppress with visual fixation or fatigue with testing; it often presents with nonotologic symptoms as well.⁷

Cranial nerve examination

Care should be taken to perform examination of cranial nerves II through XII during a complete head and neck examination for skull-based trauma. As previously mentioned, temporal bone trauma does not typically occur in isolation, and the facial trauma specialist may be the first person to identify a deficit in additional cranial nerves.

Of particular note, the petrous temporal bone comprises the anterior portion of the jugular foramen. In rare cases, a fracture can involve this structure, resulting in deficits of cranial nerves IX, X, and XI.⁸ This would present as ipsilateral weakness in shoulder raise, hoarse voice, dysphagia, and ipsilateral poor soft palate rise.⁹

Beyond an examination of cranial nerves VII and VIII, a detailed description of the rest of the cranial nerve examination is found elsewhere. An excellent review of cranial nerve anatomy and function can be found in *Cranial Nerves: In Health and Disease* by Linda Wilson-Pauwels and colleagues.¹⁰

Imaging

After a physical examination, a number of ancillary tests may be indicated to better assess the injury. A dedicated fine-cut CT of the temporal bone is almost uniformly indicated once a temporal bone fracture is identified. Temporal bone fractures have traditionally been classified as either longitudinal, meaning they go in the same direction as the petrous ridge, or transverse, meaning they are perpendicular to the petrous ridge. Fractures that involve both are considered mixed. Longitudinal fractures comprise approximately 70% to 90% of temporal bone fractures, whereas 10% to 30% are transverse.¹¹

The most important radiographic finding, however, is whether or not the fracture involves part of the cochlear or vestibular system.¹² These structures as a unit are referred to as the otic capsule. To reflect this, modern classification of temporal bone fractures classify fractures as only “otic capsule sparing” or “otic capsule disrupting.”^{2,12} Both facial nerve involvement and CSF leaks are closely associated with otic capsule involvement. In fact, otic capsule disrupting fractures convey a fivefold increase in facial nerve involvement and a twofold increase in CSF leaks (Figs. 8 and 9).¹¹

After identifying the fracture, find the facial nerve and follow its course through the temporal bone. The facial nerve enters the temporal bone at the distal end of the internal auditory canal. This segment, the labyrinthine segment, is the narrowest portion of the facial nerve, making it prone to injury. After running approximately 4 mm anterolaterally, the facial nerve turns approximately 70° at the geniculate ganglion, and runs in a posterolateral direction.¹³ This segment, the tympanic segment, courses posteriorly for approximately 13 mm, before descending toward the stylomastoid foramen at the second genu, at an angle of about 110°. The mastoid segment courses approximately 20 mm before exiting the skull.¹⁴ Disruptions along this course can sometimes be identified.

Care should also be taken to identify any disruptions of the tegmen, particularly if a CSF leak is suspected. Fluid in the mastoid air cells can represent CSF, perilymph, or blood.

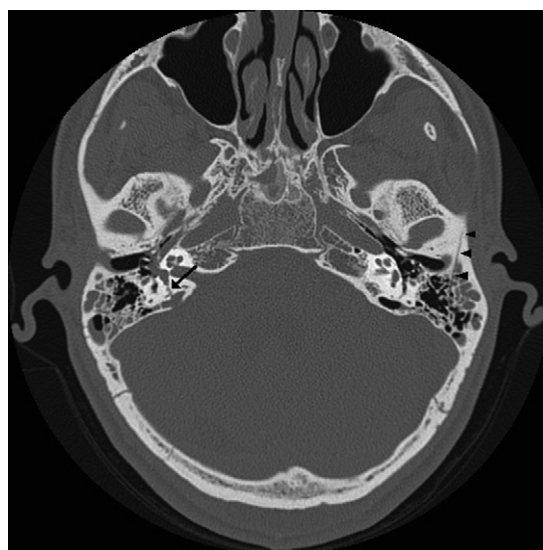


Fig. 8 The fracture on the right (*large arrow*) involves the otic capsule. Note the fracture through the patient's vestibule. There is also an otic capsule sparing fracture in the left mastoid (*arrowheads*). Note the opacification of the mastoid air cells.

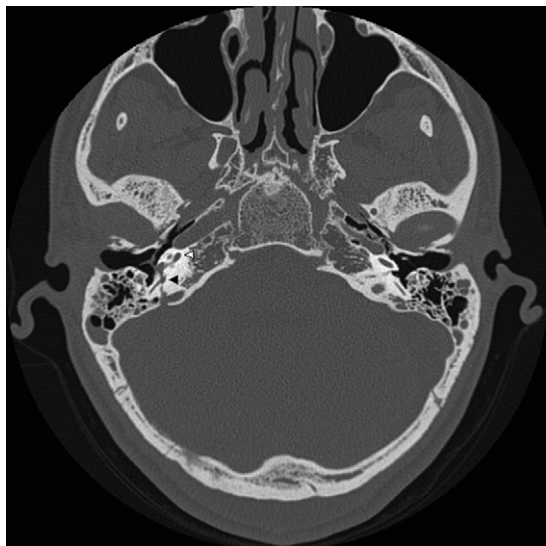


Fig. 9 This is the same patient as in Fig. 8 in a different cut. Note that the right-sided fracture travels through the cochlea (white arrowhead) and the superior semicircular canal (black arrowhead). The facial nerve was dysfunctional in this patient, and the facial nerve was graded at a House-Brackmann VI.

Fractures through the carotid canal or jugular foramen can occasionally be identified. These are more worrisome findings because of the potential impingement on cerebrovascular flow.

Management and triage

Initial evaluation of temporal bone trauma is uniform. Life-threatening emergencies are addressed first. Hemorrhage of the ear is packed. Gross CSF leaks require immediate otolaryngology and neurosurgery consults. This is followed by a thorough history and physical, as well as fine-cut CT of the temporal bone. Scalp lacerations are irrigated, debrided, and repaired, and auricular hematomas are drained and bolstered. Once this is completed, attention can be focused on the complications of temporal bone trauma.

Hearing loss

If there is concern for hearing loss, obtaining an audiogram is recommended. It should include bone conduction and tympanometry. Although the cause of conductive hearing loss can often be determined with the physical examination and sensorineural loss can be suggested from a CT demonstrating a fracture through the otic capsule, the audiogram helps identify mixed losses and quantifies the loss.

In addition, hearing loss in the presence of trauma also warrants consideration of an otolaryngology consult.

Conductive hearing loss

Conductive hearing loss in trauma is generally the result of debris obstructing the external auditory canal, fluid behind the tympanic membrane, ossicular chain discontinuity, or tympanic membrane perforation.

Debris in the ear canal can be removed easily under binocular microscopy. Irrigation of the external auditory canal is not recommended in the case of trauma, because of the possibility of a tympanic membrane perforation or communication with CSF.

Clear fluid in the ear canal suggests a CSF leak, which is discussed further later in this article.

Fluid behind the tympanic membrane is generally blood, CSF, or perilymph. Hemotympanum warrants a CT of the temporal bone, if it has not already been conducted. Hemotympanum is very common in temporal bone trauma and will resolve over the course of several weeks. After resolution, at about 6 weeks, an audiogram should be performed. Clear fluid in the middle ear space sufficient to cause a conductive hearing loss is likely CSF. A patient with a perilymphatic fistula is more likely to have sensorineural or mixed loss.

Ossicular discontinuity is usually the result of dislocation of the incudostapedial joint or of the incus itself.¹¹ It occurs in approximately 20% of temporal bone traumas.¹¹ The patient will have conductive hearing loss, and it can occur in the presence or absence of a tympanic membrane perforation. If the tympanic membrane is intact, the patient will have a maximal conductive hearing loss: a 60-db difference between the BC and AC on the audiogram. This can be fixed with ossicular chain reconstruction by an otolaryngologist.

Tympanic membrane perforations have a varied course. In the case of blast injury, 50% to 80% will spontaneously resolve.^{3,15,16} After at least 6 weeks following injury, the patient should be reexamined with a physical examination and an audiogram to determine if there is resolution. If there is no resolution, the patient should be referred to an otolaryngologist for a tympanoplasty to improve the patient's hearing and to protect the middle ear from waterborne infections. Approximately 10% of patients with tympanic membrane perforations secondary to blast have evidence of cholesteatoma.¹⁶ For those patients who obtain tympanoplasties, epithelium can be identified and removed. For those tympanic membranes that spontaneously close, this can potentially cause conductive or sensorineural hearing loss years after the initial injury. Otolaryngology consultation is warranted if there is suspicion for cholesteatoma.

Sensorineural hearing loss

Traumatic sensorineural hearing loss is typically the result of noise-induced trauma, otic capsule disruption, or perilymphic fistulas.

Noise-induced sensorineural hearing loss is a common complaint, particularly after blast injuries, and can be temporary or permanent. These tend to be in the high frequencies (2000–8000 Hz) and relatively mild.¹⁷ Temporary sensorineural hearing loss typical resolves within the first several weeks.¹⁸ Rates of permanent sensorineural hearing loss following blast injuries vary between 35% and 54%.¹⁷

Otic capsule fractures will typically cause severe to profound sensorineural hearing loss. This fracture disrupts the basilar membrane of the cochlea, the blood supply can be lost, blood can enter the cochlea, and the cochlear nerve itself can be damaged. If there is no significant resolution of hearing on repeat audiograms several months later, the patient can be offered contralateral routing of signals (CROS) hearing aids or implantable hearing devices.

Patients with perilymphatic fistulas may present with fluctuating hearing and vestibular symptoms. A typical history is a patient with Eustachian tube dysfunction who goes scuba diving and cannot hear following the dive. These can also occur as a result of trauma. If a patient has fluctuating symptoms over the course of several weeks, an otolaryngology consult should be placed for potential surgical exploration and repair of the perilymphatic fistula.

Facial nerve injury

The keys for appropriate management of facial nerve injuries are the timing of the facial nerve dysfunction and the degree of the facial nerve dysfunction. The options for management are conservative treatment with observation and high-dose steroid administration, or surgical treatment by an otolaryngologist (Fig. 10).

These injuries occur in about 7% of temporal bone fractures, and about 25% of facial nerve injuries involve complete paralysis (House-Brackmann VI).² In the largest series of temporal bone fractures to date, 27% of facial nerve injuries were immediate onset; the other 73% were delayed onset.² The time between injury and deficit ranges from 1 to 16 days.

Conservative management is appropriate in patients with delayed onset facial paralysis. In 2 larger series that explored facial nerve dysfunction following temporal bone trauma, 94% had complete recovery of function of their facial nerve without surgical intervention.^{19,20} In McKennan and Chole's¹⁹ series of 37 patients with delayed onset facial nerve dysfunction, no surgical intervention was performed and only 1 patient failed to completely recover; that patient still returned to a House-Brackmann II. In Turner's²⁰ series of 36 patients, 35 patients completely recovered, but 1 patient lost complete function of the facial nerve in the setting of an acute otitis.

Conservative management is also appropriate in patients with incomplete paralysis. Incomplete paralysis suggests that the nerve is not severed. Typically, these patients will recover completely as well.²

For those patients with immediate and complete paralysis of the facial nerve, an otolaryngologist should be consulted for possible surgical management. Typically, the site of the injured nerve in blunt trauma is the peri-geniculate region.

No matter the onset of presentation, measures should be taken to protect the eye from corneal desiccation in patients who present with a House-Brackmann VI or worse. Eye drops, such as carboxycellulose, should be administered frequently, and the eye can be taped shut at night until definitive management or resolution of symptoms occurs.

CSF leak

CSF leaks are important to identify, because of the risk of meningitis while a fistula exists. Many leaks heal on their own with conservative management alone, but larger leaks may require surgical intervention to repair the fistula. Conservative management includes elevating the head of the bed, placing the patient on strict bed rest, and starting stool softeners.¹¹

Patients with obvious CSF leaks or leaks that fail conservative management require neurosurgery and otolaryngology consultation for potential lumbar drain placement and surgical management. If there is a sufficient amount of the fluid, roughly 0.5 mL, this can be collected and sent for b2-transferrin to confirm the CSF leak. This, in conjunction with fine-cut CT imaging, is sensitive for CSF leaks.²¹ If there is an insufficient sample, or a more rapid test is required, the fluid can be sent for glucose. The glucose concentration of the fluid should be a little more than half the concentration of glucose in serum if it is CSF (40–80 mg/dL).²² If it is mucous, it will have a higher glucose concentration. This method should be used with caution because of the poor sensitivity and specificity associated with glucose and protein tests.²¹

The role of prophylactic antibiotics is somewhat controversial, because none of the reported articles has sufficient power to demonstrate their efficacy.^{2,11,23} Ratilal and colleagues²³ reviewed this topic for the Cochrane Database in

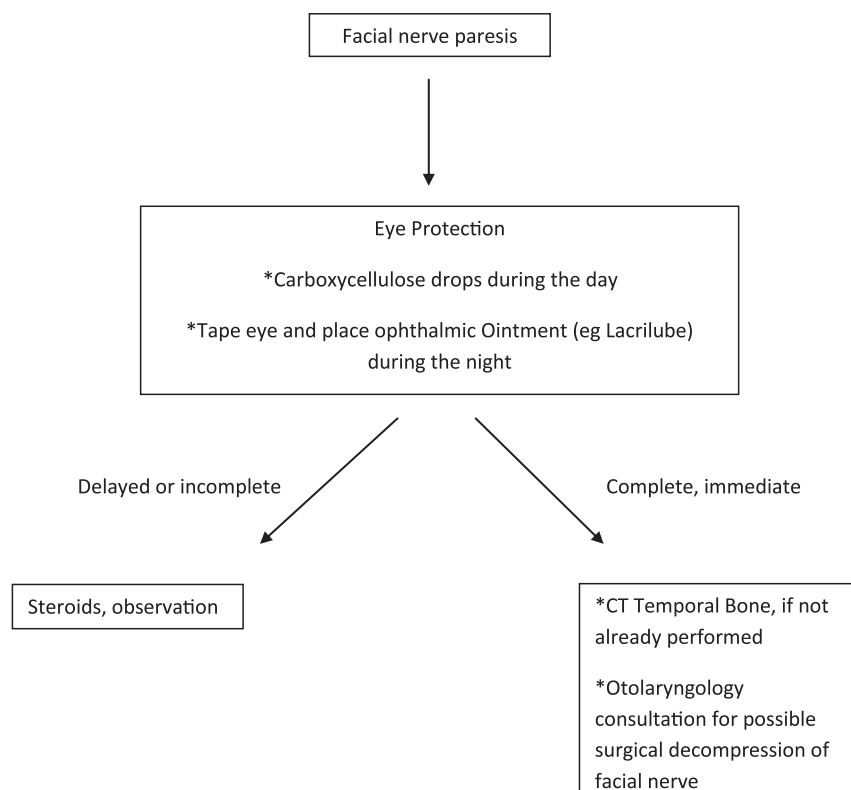


Fig. 10 An algorithm for appropriate management of facial nerve injuries.

2011, and determined that the reported evidence does not support giving prophylactic antibiotics. At the same time, posttraumatic meningitis is potentially life threatening, and antibiotics to cover the most common infecting organisms (*Streptococcus pneumoniae* and *Haemophilus influenzae*) are inexpensive.^{11,24} Many clinicians will give antibiotics on these facts alone.

Vertigo

As mentioned previously, the most common cause of dizziness in trauma is benign paroxysmal vertigo. Fortunately, this is a condition that is easily treated by an otolaryngologist with an Epley maneuver. The goal of this procedure is to reposition the dislodged otoliths and prevent them from stimulating the ampullae in the semicircular canals.

Fluctuating vertigo and hearing loss can be found in perilymphatic fistulas, as discussed previously.

Otic capsule fractures can cause permanent loss of the vestibular system of the injured ear. These patients will have severe vertigo lasting days, followed by resolution of symptoms over the next weeks as central compensation occurs.

In patients with severe vertigo, vestibular suppressants, such as scopolamine or valium, may be offered in the first few days of symptoms. Longer-term usage of vestibular suppressants, however, can interfere with compensation. Vestibular physical therapy and otolaryngology consultation is recommended in any patient with vertiginous complaints.

Auricular trauma

Hematomas of the auricle, if encountered, should be drained and bolstered. Failing to drain these may result in "cauliflower ear," which is the result of fibrinous remodeling of the cartilage from a lack of blood supply from its perichondrium. After drainage, bolsters can be easily fashioned by suturing Vaseline gauze over the drained hematoma, using 2-0 prolene. This prevents reaccumulation of the hematoma and reapproximates the perichondrium to the auricular cartilage. Care should be made not to suture the bolster in too tightly. This can cause necrosis of the thin auricular skin. At our institution, we will often place a dental roll under the suture on the posterior ear to prevent this complication. The patient should be reevaluated in a week and the bolster removed at that time.

After washing out a linear laceration through the lobule, it can be safely managed by suturing it in layers. The deep tissue should be closed with 4-0 or 5-0 Vicryl to close dead space. The skin can be closed with 4-0 or 5-0 prolene in an interrupted fashion.²⁵

Complex lacerations of the auricular, particularly those that include the cartilage, should be referred to either an otolaryngologist or a facial plastic surgeon.

Carotid artery injury

The carotid canal lies next to the Eustachian tube and can be injured in temporal bone fractures. These injuries are rare, occurring in roughly 1% of severe temporal bone trauma, but they are life threatening.² If there is hemorrhagic otorrhea, the ear should be packed to prevent continued bleeding, and an emergent consult to neurosurgery should be placed. An emergent arteriogram with possible embolization by neurosurgery should be considered if the neurologic examination is not consistent with the CT scan, a fracture can be seen through the carotid canal on the CT, or there are lateralizing neurologic deficits.²⁶

Gunshot wound

Gunshot wounds have variable presentations, but the algorithm is largely the same. Arterial bleeding should be controlled immediately. If there is herniation of brain into the wound, emergent otolaryngology and neurosurgery consultations should be made.

After resolution of emergent issues, the mastoid and ear are often obliterated to prevent traumatic implantation of cholesteatoma.²⁶

External auditory canal fracture

Fractures of the external auditory canal should be followed on a long-term basis. Although rare, these patients risk canal wall cholesteatomas growing into the fracture line, and they risk external auditory canal stenosis, particularly if there is disruption of the skin of the external auditory canal.¹¹

References

1. Travis LW, Stalnaker RL, Melvin JW. Impact trauma of the human temporal bone. *J Trauma* 1977;17(10):761–6.
2. Brodie HA, Thompson TC. Management of complications from 820 temporal bone fractures. *Am J Otol* 1997;18(2):188–97.
3. Ritenour AE, Wickley A, Ritenour JS, et al. Tympanic membrane perforation and hearing loss from blast overpressure in Operation Enduring Freedom and Operation Iraqi Freedom wounded. *J Trauma* 2008;64(Suppl 2):S174–8 [discussion: S178].
4. Kang TS, Vrabec JT, Giddings N, et al. Facial nerve grading systems (1985–2002): beyond the House-Brackmann scale. *Otol Neurotol* 2002;23(5):767–71.
5. House JW, Brackmann DE. Facial nerve grading system. *Otolaryngol Head Neck Surg* 1985;93(2):146–7.
6. Lee SH, Kim JS. Benign paroxysmal positional vertigo. *J Clin Neurol* 2010;6(2):51–63.
7. Traccis S, Zoroddu GF, Zecca MT, et al. Evaluating patients with vertigo: bedside examination. *Neurol Sci* 2004;25(Suppl 1):S16–9.
8. Yildirim A, Gurelik M, Gumus C, et al. Fracture of skull base with delayed multiple cranial nerve palsies. *Pediatr Emerg Care* 2005;21(7):440–2.
9. Crue BL, Freshwater DB, Shelden CH, et al. Syndrome of the jugular foramen. *AMA Arch Otolaryngol* 1956;63(4):384–91.
10. Wilson-Pauwels L, Akesson EJ, Stewart PA, et al. Cranial nerves: in health and disease. 2nd edition. Hamilton (Ontario): BC Decker, Inc; 2002.
11. Brodie HA. Management of temporal bone trauma. In: Flint PW, editor. *Cummings otolaryngology: head & neck surgery*. 5th edition, vol. 2. Philadelphia: Mosby; 2010. p. 2036–48.
12. Ishman SL, Friedland DR. Temporal bone fractures: traditional classification and clinical relevance. *Laryngoscope* 2004;114(10):1734–41.
13. Jager L, Reiser M. CT and MR imaging of the normal and pathologic conditions of the facial nerve. *Eur J Radiol* 2001;40(2):133–46.
14. Phillips CD, Bubash LA. The facial nerve: anatomy and common pathology. *Semin Ultrasound CT MR* 2002;23(3):202–17.
15. Chait RH, Casler J, Zajtcuk JT. Blast injury of the ear: historical perspective. *Ann Otol Rhinol Laryngol Suppl* 1989;140:9–12.
16. Sridhara SK, Rivera A, Littlefield P. Tympanoplasty for blast-induced perforations: the Walter Reed experience. *Otolaryngol Head Neck Surg* 2013;148(1):103–7.
17. Cave KM, Cornish EM, Chandler DW. Blast injury of the ear: clinical update from the global war on terror. *Mil Med* 2007;172(7):726–30.
18. Cohen JT, Ziv G, Bloom J, et al. Blast injury of the ear in a confined space explosion: auditory and vestibular evaluation. *Isr Med Assoc J* 2002;4(7):559–62.
19. McKennan KX, Chole RA. Facial paralysis in temporal bone trauma. *Am J Otol* 1992;13(2):167–72.

20. Turner JW. Facial palsy in closed head injuries the prognosis. *Lancet* 1944;243(6302):756–7.
21. Chan DT, Poon WS, Ip CP, et al. How useful is glucose detection in diagnosing cerebrospinal fluid leak? The rational use of CT and Beta-2 transferrin assay in detection of cerebrospinal fluid fistula. *Asian J Surg* 2004;27(1):39–42.
22. McGuirt Jr WF, Stool SE. Cerebrospinal fluid fistula: the identification and management in pediatric temporal bone fractures. *Laryngoscope* 1995;105(4 Pt 1):359–64.
23. Ratilal BO, Costa J, Sampaio C, et al. Antibiotic prophylaxis for preventing meningitis in patients with basilar skull fractures. *Cochrane Database Syst Rev* 2011;(8). CD004884.
24. Eftekhari B, Ghodsi M, Hadadi A, et al. Prophylactic antibiotic for prevention of posttraumatic meningitis after traumatic pneumocephalus: design and rationale of a placebo-controlled randomized multicenter trial [ISRCTN71132784]. *Trials* 2006;7:2.
25. Marcus BC. Wound closure techniques. In: Baker SR, editor. *Baker: local flaps in facial reconstruction*. 2nd edition. China: Mosby; 2007. p. 41–64.
26. Shindo ML, Fetterman BL, Shih L, et al. Gunshot wounds of the temporal bone: a rational approach to evaluation and management. *Otolaryngol Head Neck Surg* 1995;112(4):533–9.

Further readings

- Brodie HA, Thompson TC. Management of complications from 820 temporal bone fractures. *Am J Otol* 1997;18(2):188–97.
- Brodie HA. Management of temporal bone trauma. In: Flint PW, editor. *Cummings otolaryngology: head & neck surgery*. 5th edition, vol. 2. Philadelphia: Mosby; 2010. p. 2036–48.
- Phillips CD, Bubash LA. The facial nerve: anatomy and common pathology. *Semin Ultrasound CT MR* 2002;23(3):202–17.
- Sridhara SK, Rivera A, Littlefield P. Tympanoplasty for blast-induced perforations: the Walter Reed experience. *Otolaryngol Head Neck Surg* 2013;148(1):103–7.
- Wilson-Pauwels L, Akesson EJ, Stewart PA, et al. *Cranial nerves: in health and disease*. 2nd edition. Hamilton (Ontario): BC Decker, Inc; 2002.

Reconstruction of Hard and Soft Tissue Maxillofacial Defects

Christopher M. Harris, DMD, MD ^{a,*}, Robert Laughlin, DMD ^b

KEYWORDS

• Reconstruction • Maxillofacial surgery • Soft tissue • Hard tissue

KEY POINTS

- Reconstruction of maxillofacial composite defects is a technically demanding and time-demanding process.
- Reconstruction requires a prolonged treatment course, a team approach, and meticulous planning that is prosthetic and esthetically driven.
- The use of vascularized flap reconstruction, dental implants, and computer-aided technology and advances in maxillofacial prosthetics have contributed immensely toward the goal of fully reconstructing victims of large avulsive wounds.
- Further advances in technology, surgical training, and maxillofacial prosthodontics will undoubtedly aid in minimizing the number of surgical interventions and maximize the final functional and esthetic results of these patients.

Introduction

There are multiple methods to reconstruct large hard and soft tissue defects, as well as composite defects. There is no one panacea reconstructive option for every defect and each technique has advantages and disadvantages. In select cases, vascularized free flap reconstruction is the ideal choice; in others, local flaps and secondary bone grafting can provide equivalent, or superior, functional and esthetic results. Multiple algorithms have been presented in the literature to address the various defects and their reconstruction. The purpose of this article is to provide a general overview of the authors' insights into the surgical and prosthetic management of hard and soft tissue defects. Discussed are some of the more common complications that the authors have encountered during the course of treatment and the suggested management.

The management of hard, soft, and composite tissue defects of the maxillofacial region can present multiple issues of varying complexity along the treatment timeline. Composite injuries involve the disruption, or loss, of multiple tissue types that typically result from traumatic injuries or ablative surgical procedures. The reconstruction of these wounds requires

replacement of multiple tissue types and must also reconstitute form and function. The surgeon must realize that facial esthetic units are functional and 3-dimensional and consist of multiple superficial and deeper supporting components. Unfortunately, some patients are managed with inadequate procedures that lengthen the treatment time or adversely affect final esthetic and functional results. The surgeon must be cognizant of the goal of their reconstructive efforts and their ability to manage it. This recognition should begin at the initial intervention. In many maxillofacial wounds, restoration of a functional and esthetic dentition via maxillofacial prosthodontics is the endpoint of therapy. Some of the issues surrounding the reconstruction of composite defects include the following:

- Minimization of surgical procedures
- More rapid definitive reconstruction
- Three-dimensional, prosthetic-driven treatment planning
- Achievement of bone continuity
- Alveolar reconstruction
- Adequate soft tissue replacement
- Facial soft tissue esthetics
- Vascularized versus nonvascularized tissue reconstruction
- Dental implants and prosthetics
- Management of secondary complications

The authors' approach to these complex cases is to address the reconstruction in 3 basic phases: soft tissue (facial and intraoral), supporting hard tissues, and dentoalveolar (alveolar and dental) reconstruction and secondary procedures. This approach ideally yields the minimum number of procedures per phase to accomplish the primary goal of that phase. This approach decreases the number of procedures, healing time, and total treatment time. Ideally, the replacement all the tissues needed for reconstruction occurs in 1 phase. With the advent of delayed primary reconstruction with vascularized composite tissue transfer and computed tomography (CT)-guided surgical templates directing bone and dental implant reconstruction, this

Disclaimer: The views expressed in this article are those of the author(s) and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government.

We are military service members. This work was prepared as part of our official duties. Title 17 U.S.C. 105 provides that 'Copyright protection under this title is not available for any work of the United States Government.' Title 17 U.S.C. 101 defines a United States Government work as a work prepared by a military service member or employee of the United States Government as part of that person's official duties.

^aOral and Maxillofacial Surgery, Naval Medical Center Portsmouth, 620 John Paul Jones Circle, Portsmouth, VA 23708, USA

^bOral and Maxillofacial Surgery, Naval Medical Center San Diego, 34800 Bob Wilson Drive, San Diego, CA 92134, USA

* Corresponding author.

E-mail address: Christopher.Harris4@med.navy.mil

is becoming a reality. However, in many cases, due to lack of facilities, finances, technology, or training, this is not possible. The authors think that this combination of technology and surgery, providing single-stage, multiphase reconstruction, will become the standard of care in the future.

Evaluation and management of the wound

Gunshot wounds and blast injuries on initial presentation are contaminated, unstable, and evolving wounds. Wounds caused by ablative surgery are typically planned, stable defects. Traumatic avulsive injuries require initial stabilization of the patient and the focus is on the management of issues pertaining to the patient's survival. Establishment of a secure airway and management of life-threatening wounds take precedence over maxillofacial injuries no matter what their appearance is (Box 1).

Once these acute issues are addressed, wound debridement, structural stabilization, and interventions to reduce infection and tissue loss are performed. In patients undergoing ablative surgery, these acute issues are not a large factor because the reconstruction is planned for preoperatively. However, once traumatic wounds have declared themselves and are clean and healthy and the defect size and type are well defined, the approach to reconstruction is quite similar. Basically, it involves categorizing the missing or unusable tissue (ie, severely scarred tissue) and devising a comprehensive treatment plan to accomplish the replacement. The evaluation and management of acute maxillofacial wounds are covered comprehensively elsewhere in the literature.

The classic management of avulsive wounds, such as gunshot wounds, is to make every attempt to stabilize the wound and then perform delayed repair of missing hard and

scarred soft tissues. These techniques include external fixation or internal fixation, minimal debridement, and closure of intraoral and extraoral wounds. Resultant scarring and wound contraction make secondary reconstruction difficult at best with these techniques.

Experience gained from the Global War on Terror and large urban trauma centers supports a newer approach, using transitional plating techniques and bone grafting to stent soft tissue against contracture until a healthy wound is realized. At this point, a delayed, definitive reconstruction is undertaken. These protocols were introduced to address the multiple issues surrounding these wounds, such as potentially massive multi-system trauma, grossly contaminated or colonized wounds, high-energy gunshot, burn, and blast injuries, and the frequent prolonged time from initial injury to definitive care. Although these types of injuries are not commonly encountered by non-US military practitioners, composite defects, whether they be ablative or traumatic in nature, are not uncommon. Orthopedic and plastic surgeons have been using vascularized regional and free tissue flaps for nearly 20 years in the reconstructive management of traumatic extremity and burn wounds. Civilian trauma centers have documented the benefits of a delayed, primary reconstruction of avulsive maxillofacial injuries for more than a decade.

Presurgical treatment planning

Team approach

A team-centered treatment plan for the management of composite wounds is essential to achieve the best final outcome. This team should consist minimally of the reconstructive surgeon and a maxillofacial prosthodontist. In addition, recruitment of other surgical services to provide for soft tissue, hard tissue, or composite tissue transfer may be required. The wound care team, psychiatry, speech therapy, and physical and occupational therapy may also be needed.

Imaging in reconstruction

CT scanning is the gold standard for the evaluation and planning for these injuries. Plain films have a limited role in evaluating these patients. Before any intervention, a thorough clinical examination should be performed. Radiology data may not show occult soft tissue injuries requiring prompt attention before the definitive reconstruction (eg, ocular, salivary gland, or facial nerve injuries). CT data provide for detailed planning, accurate measurements, and the production of stereolithographic models. The authors routinely use these for preoperative reconstruction plate shaping and screw depth determination.

CT data also allow for the use of intraoperative surgical navigation and computer-aided design/computer-aided manufacturing (CAD/CAM)-based ablative and reconstructive surgical guides. At the authors' facility, with the treatment planning of vascularized fibula free flap cases, CT angiography of the lower extremities is routinely used for the evaluation of vessels, but also for CT data to construct CAD/CAM cutting guides, which allows for an in situ shaping of the fibula while attached to the vascular pedicle. In the cases in which the authors have used this, minimal additional reshaping is needed before flap inset. The treatment planning, ischemic time, and overall operative time saved with this technology is well worth the expense (Figs. 1–4).

Box 1. Management of avulsive traumatic wounds

1. Initial stabilization of patient medically and surgically
2. Clinical examination to identify injuries, particularly globe, soft tissue, and dental injuries
3. Obtain initial computed tomography scans
4. Obtain wound cultures and sensitivities if indicated
5. Scheduled, serial wound washouts and debridement until wound is healthy and stable
6. Obtain additional imaging; consider stereolithographic model or computer-aided design/computer-aided manufacturing surgical guides
7. Stabilize hard tissues to support soft tissues and decrease wound contracture
8. Consider free vascularized flap reconstruction for delayed primary reconstruction for composite wounds, or
9. Consider vascularized flap to replace and augment missing soft tissue
10. Perform definitive bone reconstruction (if no composite tissue flap used)
11. Dentoalveolar reconstruction (eg, implants, interim prosthetics)
12. Secondary procedures during interim prosthetic period (eg, vestibuloplasty, flap debulking, gingival grafts)
13. Deliver final prosthetic; perform cosmetic revisions



Fig. 1 Example of the CAD/CAM produced cutting guides for the mandible and fibula and stereolithographic model for preoperative plate bending.

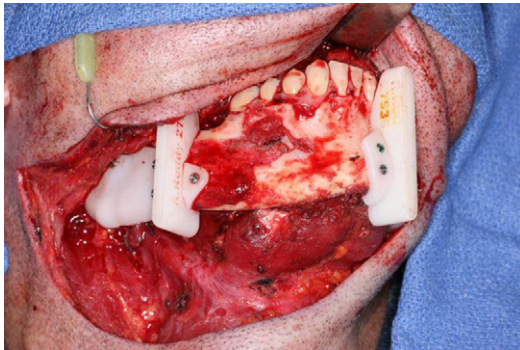


Fig. 2 Mandibular resection guide.

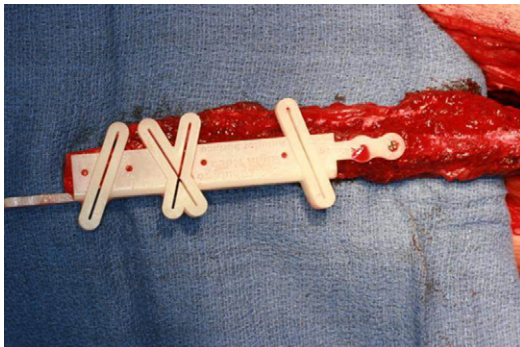


Fig. 3 Fibula shaping guide.



Fig. 4 Postoperative panoramic radiograph.

Vascularized tissue transfer

Many oral and maxillofacial surgeons do not have vascularized composite tissue transfer as part of their armamentarium. It should be noted that these techniques may not be the best reconstructive procedure in every case. Excellent reconstructive outcomes can, and have been, delivered with multiply staged procedures. However, recognition that a single-procedure, free tissue composite tissue transfer may be the most appropriate method to reconstruct some complex avulsive defects is essential. Lack of surgical training to perform these procedures should not deprive the maxillofacial reconstruction patient the potential option for a single-procedure, yet multiphase, surgical reconstruction. Appropriate consultation with other surgical specialties, meticulous preoperative planning, and accurate intraoperative placement of bone flaps by the oral and maxillofacial surgeon is paramount. The primary role of oral and maxillofacial surgeons who do not perform these tissue transfers is to ensure the correct, preplanned bone flap inset and soft tissue closure. Bone-containing flaps that obtain continuity yet are placed in nonanatomic, unusable positions for dental implant placement can significantly undermine the final dental prosthetic reconstruction and may be nonrestorable. These errors may require another surgical procedure to reconstruct the prior reconstruction.

Soft tissue reconstruction

Replacement of soft tissue is required for a good functional and cosmetic result. Cosmetically, it is preferred to use local soft tissue flaps for facial soft tissue defects, because they most closely match surrounding tissue characteristics. Vascularized soft tissue flaps (ie, free tissue transfer and regional flaps) can also replace missing facial soft tissue, but can be unesthetic due to differences with local tissue characteristics (Figs. 5–7). Some large area and large volume soft tissue defects can only be adequately addressed with vascularized free flaps or regional flaps. When addressing composite wounds, local soft tissue flaps frequently lack the volume and size needed to support the second bone grafting phase of reconstruction. It is paramount that a composite defect has an adequate volume of healthy soft tissue. A presence of a low volume, scarred soft tissue wound bed will dramatically increase complications and limit the required volume needed for secondary bone grafting (Figs. 8–11).

The most commonly used soft tissue reconstructive modalities in the authors' practice are the various cervical and facial flaps, skin grafting, buccal fat pad flaps, pectoralis major flap, and the vascularized free radial forearm and anterolateral



Fig. 5 Radial forearm-palmaris longus flap for lip reconstruction. Poor soft tissue match with native facial skin.

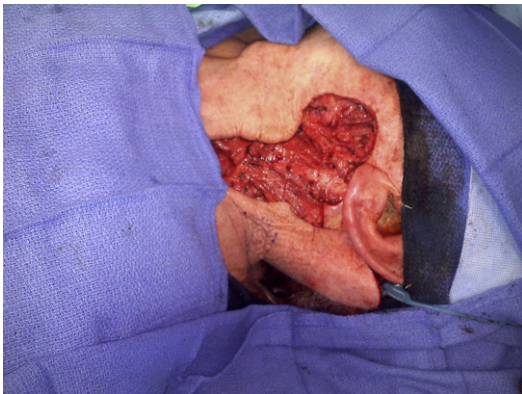


Fig. 6 Recurrent squamous cell carcinoma (SCCA) cheek defect.



Fig. 7 Inset of flap. Improved facial tissue match resulting from local tissue reconstruction.



Fig. 8 Frontal photo of gunshot wound (GSW) to lower face and mandible.

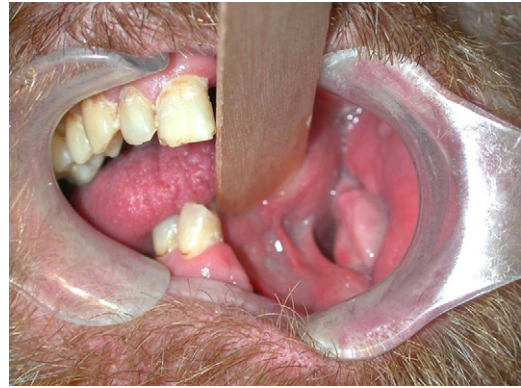


Fig. 9 Intraoral photo of GSW to lower face and mandible. Note large composite tissue loss.



Fig. 10 Panoramic radiograph of mandibular defect.

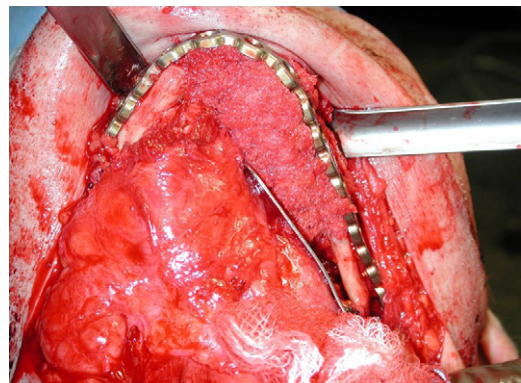


Fig. 11 Bone reconstruction without soft tissue (extraoral and intraoral) reconstruction.

Box 2. Complications commonly seen with soft tissue reconstruction

1. Wound infection or dehiscence
2. Partial or total flap loss
3. Wound and flap contraction
4. Dissimilar tissue characteristics (color, texture, hair-bearing, thickness)
5. Osseointegrated implant-skin interface inflammation

thigh flaps. The anatomy and harvest techniques of these flaps are available elsewhere in the literature and are not discussed here. The reconstructive surgeon should be technically proficient enough to perform most of these procedures. At a minimum, the surgeon must have competence using large regional skin and musculocutaneous flaps before attempting a large composite defect reconstruction (Box 2).

Infection and flap loss

Wound infection, flap loss, and subsequent wound contraction are rarely considered when planning the initial soft tissue reconstructive plan. Unfortunately, they do occur and can have significant impact on the final soft tissue esthetic and functional results. Infection and partial/total flap loss necessitate wound debridement and removal of needed tissue components. Additional local flaps and grafting procedures may have to be used to maximize the now deficient soft tissue reconstruction. Scarring, wound contraction, and the resultant decrease in vascularity can adversely affect the secondary bone grafting or prosthetic plan.

Ensuring a healthy wound before reconstruction, appropriate perioperative antibiotic protocols, sterile perioperative wound care, and optimization of the patient's nutritional status are imperative before soft tissue reconstruction procedures. Maintenance and optimization of nutritional status are typically undertaken with nasogastric or polyethylene glycol tube enteral feedings. The wound care services in most hospitals can provide excellent assistance with daily wound care and debridement, wound care materials, as well as VAC therapy in required cases. In most cases, these adjunctive procedures and meticulous surgical management can minimize the negative outcomes of infection and partial flap loss.

Total flap loss will require flap removal and debridement as well as a secondary reconstructive procedure. Although vascularized free flaps have a rate of success approaching 95% in most institutions, the typical pattern of failure is total flap loss. This all or none failure pattern is one of the negative aspects of vascularized free flaps reconstruction. However, the rarity of vascularized free flap failure does decrease the postoperative issues more commonly seen with partial flap loss in large cutaneous and regional myocutaneous flaps (Figs. 12–17).

Dissimilar tissue characteristics

Tissue characteristics are important primarily for esthetic results when replacing facial tissue with distant tissue. However, tissue thickness can also affect functional aspects of the reconstruction as well. Excess tissue bulk is commonly seen after the soft tissue reconstruction, but over several weeks to months, this bulk typically decreases. Regional flaps and



Fig. 12 Tissue breakdown due to poor soft tissue quality.



Fig. 13 Full-thickness cheek wound related to buccal mucosa and lip SCCA.



Fig. 14 Intraoperative ischemic electrocardiographic changes required abortion of planned radial forearm free flap (RFFF). Buccal fat pad advancement and cervicofacial advancement flap provided 2-layer closure of defect.

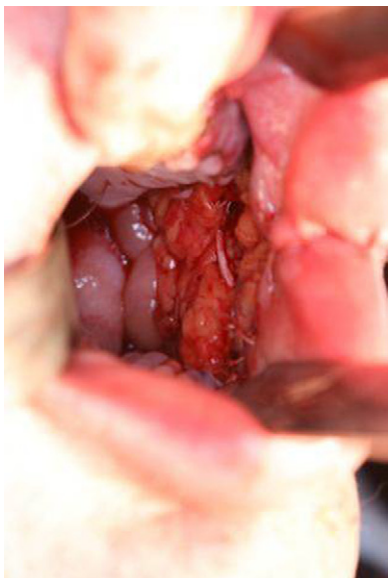


Fig. 15 Intraoral view of buccal fat pad (BFP) advancement flap.



Fig. 16 Postoperative tracheostomy and flap infection resulted in loss of flap periphery.



Fig. 17 Debridement and wound vacuum assisted closure (VAC) therapy left area requiring additional full-thickness grafting.



Fig. 18 Final result of full-thickness defect reconstruction. Resultant scarring left anterior maxillary and mandibular implants inaccessible for maxillofacial prosthodontist.

vascularized free flaps with large muscle components will undergo significant atrophy (see Figs. 15 and 16). With osteocutaneous free flap reconstructions, serial tissue debulking can be performed to decrease the skin surface distance from underlying bone in an attempt to re-create the vestibule and to aid in prosthetic cleansability.

Color, skin thickness, coarseness, and presence of hair may all be problematic to patients. Frequently, intraoral skin flaps still produce hair, which can be disconcerting to the patient (see Fig. 17). These intraoral skin flaps can be managed with benign neglect (as many will decrease hair production with prolonged oral exposure), with periodic trimming, or by surface ablation. Laser hair removal of accessible skin can also be performed. Hair growth will cease in the patient receiving radiation therapy after the flap procedure for oncologic disease processes (Fig. 18).

Dental implants and the abutments react poorly to a thick skin-implant interface. Frequently, the cause is excessive thickness and lack of attached tissue, which prevents adequate cleansing (Figs. 19 and 20). Serial flap debulking or removal of the skin component and resurfacing with an allograft or allowing the wound to granulate for mucosal coverage can be performed. The use of intraoral osteofascial flaps instead of osteocutaneous flap can also decrease issues associated with soft tissue thickness; however, it typically results in the formation of unattached mucosa. Many will still require



Fig. 19 Immediate postoperative view of bulky soft tissue in hemimaxilla reconstruction with fibula osteocutaneous vascularized flap.



Fig. 20 Intraoral view of same soft tissue component 4 months postoperatively.

keratinized tissue grafting for peri-implant tissue health and for vestibular re-creation as a secondary procedure (Fig. 21).

Hard tissue reconstruction

Replacement of supporting hard tissue is essential for esthetics and function in maxillofacial reconstruction. The goals are for bone continuity and the creation of an acceptable platform for dental implants and prosthetic reconstruction. Bone continuity is the primary focus of reconstruction. In the authors' practices, implant-supported prosthodontic reconstruction is performed in most patients. However, a large portion of patients in civilian treatment facilities do not receive these procedures primarily because of the expense. Reconstruction plates and prosthetics also play an esthetic role in the replacement of underlying facial and alveolar form and with soft tissue support.

Provided a healthy wound with adequate soft tissue is available, reconstruction of the nonalveolar mandible for continuity is relatively straightforward and reliable. It can be accomplished with nonvascularized and vascularized flap techniques with good rates of success. The authors routinely perform nonvascularized bone grafting for large mandibular continuity defects with little or no soft tissue deficits with high rates of success. Large avulsive defects of the maxilla are typically reconstructed with composite tissue transfer, whereas smaller defects are reconstructed with a maxillary obturator (Figs. 22–28). It is the authors' opinion that all large composite defects from malignancies and anterior mandibular defects (benign, malignant, or traumatic) are best treated by vascularized tissue flaps.



Fig. 21 Postoperative view of skin paddle from fibula osteocutaneous vascularized flap.



Fig. 22 After laser hair removal of palate defect reconstructed with radial forearm vascularized flap.

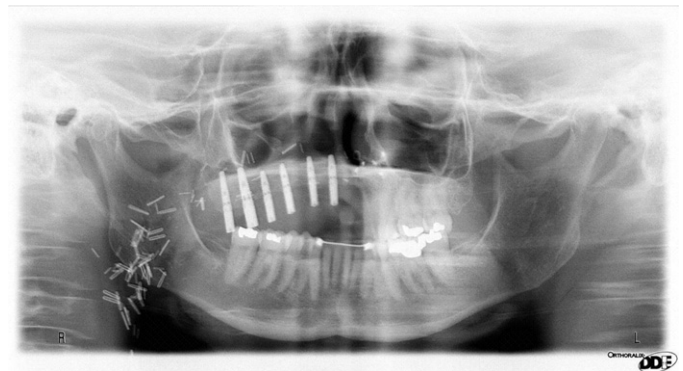


Fig. 23 Long abutments on implants placed into fibula osteocutaneous vascularized flap for hemimaxilla reconstruction.

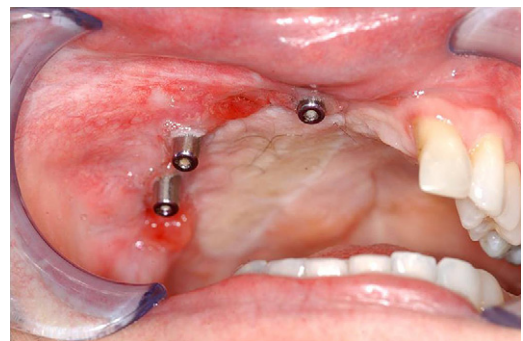


Fig. 24 Intraoral view of inflamed peri-implant tissue.



Fig. 25 Split thickness skin graft (STSG) vestibuloplasty to re-create vestibule and address peri-implant tissue inflammation.



Fig. 26 Resection specimen of a large mandibular myxoma.

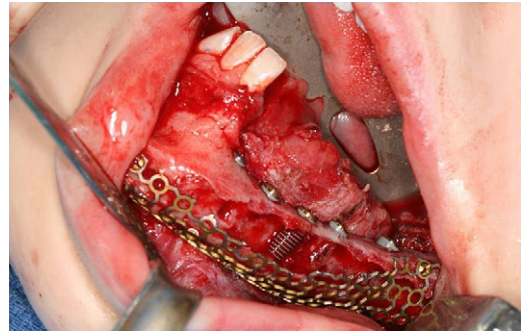


Fig. 29 Intraoral view of idealized implant placement. Buccal defect was regrafted with titanium mesh, allograft, and rh-BMP.

Alveolar reconstruction

A common issue in the reconstruction of hard tissue defects is the reconstruction of alveolar bone height. Clinically and radiographically, this lack of alveolar bone can leave a large vertical discrepancy between the native bone and the reconstructed bone. This issue is seen with nonvascularized and vascularized flap reconstruction (Figs. 29 and 30). When using nonvascularized grafting techniques, various attempts to improve alveolar height using containment with rib grafts, plates, titanium mesh, autogenous corticocancellous blocks, and periosteal tent-pole techniques with implant placement have all been described in the literature. Superior placement of bone flaps, secondary distraction osteogenesis, and double-barrel techniques have been described to address the issue when using the bone containing vascularized flaps. No single technique seems to be able to universally reconstruct alveolar height at the time of primary bone reconstruction, particularly in large defects. In reality, this alveolar discrepancy is generally reconstructed by the final prosthetic framework. This alveolar height discrepancy and the role of the prosthesis raise issues in regard to the biomechanics of this elevated prosthetic platform and implant maintenance.

Periodontal bone loss and gingival recession

Another commonly seen issue with composite resections is periodontal bone loss, root exposure, and unesthetic gingival recession on teeth adjacent to the defect (see Figs. 16, 20, and 27). Frequently this is due to the traumatic injury or ablative procedure disrupting the tooth's supporting periodontium at the time of repair and resection. Surgeons also tend



Fig. 27 Panoramic radiograph of defect and reconstruction plate.



Fig. 28 Cone beam computed tomography scan (CBCT) scan of PICBG and rh-BMP reconstruction 4 months after grafting.

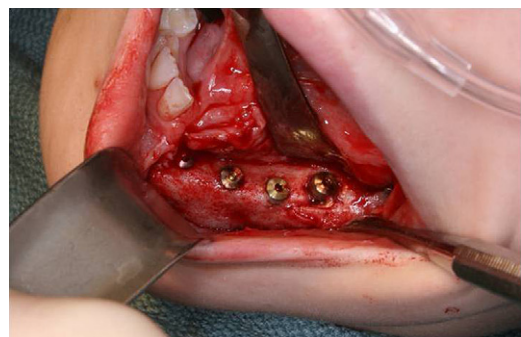


Fig. 30 Appearance of implant uncovered 4 months after regrafting procedure.

to save teeth with postinjury bone loss or coronal root exposure adjacent to the defect. In many cases, these teeth are poor abutments for a transitional, removable prosthesis (eg, maxillary obturator) and have a poor long-term periodontal prognosis. If fixed implant-supported prosthetics are planned, the authors typically remove these teeth in favor of non-compromised teeth and leave the width of a full tooth socket from the resection edge to maintain the adjacent tooth's bone level (see Figs. 29 and 30). Future implants and prosthetics are then used to replace these teeth. The authors have found a superior esthetic result, less long-term periodontal bone loss, and a reduction in unesthetic gingival recession in teeth adjacent to the prosthesis if these compromised teeth are electively sacrificed.

Nonvascularized grafts versus vascularized flaps

Mandibular reconstruction

The anterior or posterior iliac crests remain the primary sites of bone harvest for reconstructive surgeons, which is primarily due to the available bone stock available, the relative ease of harvest, the low rate of complications, and the low morbidity associated with them. The major downside of nonvascularized bone reconstruction is the unpredictable amount of bone resorption, especially in a large defect (>6 cm), deficient soft tissue base, or vascular compromise (eg, heavily scarred or radiated tissue). Extensive bone grafting, as mentioned earlier, must be supported by an ample, healthy soft tissue to ameliorate this effect. This extensive bone grafting mandates an additional major surgical procedure if vascularized composite tissue flap is not used.

Newer materials, such as rh-BMP-2, also have a seemingly positive effect on hard tissue reconstruction. In the authors' clinical experience, the addition of rh-BMP-2 to allografts and autogenous bone has allowed for better bone volume maintenance and maturation, even with large defects. In the authors' clinic, dental implants are routinely being performed 4 months after grafting when rh-BMP-2 is added to iliac crest grafts for large mandibular defects with minimal resorption. The major drawback of using the material is the expense and the significant postoperative edema seen with its use (Figs. 31–34).

In the authors' practices, the vascularized fibula free flap (bone only and composite) is the primary flap used when considering bone reconstruction of the mandible and maxilla. The fibula has an excellent available bone length, adequate bone height for most mandibular and maxillary continuity defects, and an available soft tissue component and allows for a second team to perform flap harvesting. The ample pedicle length also allows for maxillary reconstruction using the facial or superficial temporal vessels typically without the use of vein grafting.

In mandibular reconstruction, the authors typically reserve fibula free flaps for defects greater than 6 cm, all but the smallest anterior defects and those that also require soft tissue replacement (ie, malignancy or avulsive composite defects). The fibula is used in larger composite maxillary defects, because smaller defects can be easily reconstructed with a maxillary obturator or local bone and soft tissue reconstruction without extensive surgery.

Maxillary reconstruction

Soft tissue local, regional, and vascularized flaps can all be used for the obturation of maxillary defects. Using these soft tissue only techniques instead of the more appropriate



Fig. 31 Panoramic radiograph with final zirconia-based prosthesis in place. Note periodontal bone loss of adjacent mandibular incisors.



Fig. 32 Intraoral view of final prosthesis and occlusion.



Fig. 33 Panoramic radiograph of alveolar and graft vertical discrepancy in secondary posterior iliac crest bone graft (PICBG) reconstruction.

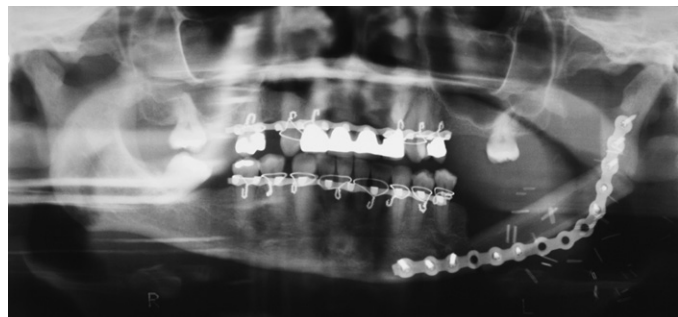


Fig. 34 Panoramic radiograph of alveolar and graft vertical discrepancy in fibula free flap primary reconstruction.

composite tissue flaps for infrastructure maxillary reconstruction prevents the use of a maxillary obturator. In dentate patients, maxillary obturation is a reliable means of addressing most maxillary functional and esthetic defects without requiring patients to undergo extensive surgical procedures. In patients with malignancy it also allows for visual inspection of the area. In edentulous patients or those with larger defects, maxillary obturation becomes more difficult and patients are generally unsatisfied with the results of the reconstruction. In these patients, local bone grafting and dental implants can be used to support a maxillary obturator, or a composite tissue flap with implant supported prosthetics should be used (Figs. 35–37).

Dentoalveolar reconstruction

The goal of the maxillofacial reconstruction phase is to recreate a biomechanically stable, functional, and esthetic result. In most cases, it is an analogue for the deficient alveolar bone structure. Early in the treatment planning process, the authors involve the maxillofacial prosthodontists in their clinic. Whether the defect cause is traumatic or ablative, the maxillofacial prosthodontists are intimately involved in the reconstruction planning phase to idealize the proper hard and soft tissue platform, ensuring normal jaw relations, and minimizing occlusal disharmony. Poor communication with these practitioners frequently leads to poor long-term results for the patient because of compromises in prosthetic design, function, secondary soft tissue issues, and overall esthetics.

In the authors' practice, dental implants are always placed with the guidance of the maxillofacial prosthodontists. Routinely, surgical guides are rarely used because the prosthodontist accompanies the authors to the operating room for direct input in regard to implant position. Surgical guides can be used if direct input is not available. Traditional guides based on dental casts alone tend to be inaccurate in regard to underlying bone position; thus guides based on CT scans with custom guides are highly recommended if the prosthodontist cannot be present. After implant placement, a bone level impression and jaw relations record are made by the prosthodontist in the operating room. This impression and record are used to manufacture an interim prosthetic for the patient.

In the authors' practice, the typical time for non-vascularized bone graft reconstruction is approximately 1.5 to



Fig. 35 Hemimaxillectomy defect and radiated field due to maxillary SCCA. A radial forearm flap performed by another surgeon to address the patient complaint of poorly fitting obturator and leakage. Soft tissue flap prevented obturator use.



Fig. 36 Patient after anterior iliac crest bone graft (AICBG) with rh-BMP grafting and implants to support dental rehabilitation.

2 years from initial surgery until the final prosthesis is delivered. With the authors' vascularized flap reconstruction, this time is approximately 6 to 9 months shorter. The difference lies primarily with the extended time requirement for non-vascularized bone graft healing and implant osseointegration in these grafts. Typically in fibula free flap reconstructions, dental implants are placed with enough primary stability to provide an interim prosthesis within a month of surgery. The authors have seen no issues with implant failure with this loading protocol with the authors' vascularized flaps. At this stage, the patients have been at least partially edentulous for a lengthy period of time and the delivery of an interim prosthetic greatly improves their morale and body image.

With the mounted dental models, the patient's casts are modified and denture teeth are set. A wax try is then performed by the prosthodontist. Once this is adjusted and finalized, the wax try-in is sent to a dental laboratory where a replica interim prosthesis is made from polyether ether ketone in a CAD/CAM designed milling process. The final product is a tooth-colored, screw-retained, fixed temporary prosthetic. This prosthetic is then delivered and the occlusion, phonetics, and esthetics are evaluated.

The patient typically wears this prosthesis for several months and has multiple visits for reevaluation and adjustments. During this period, secondary soft tissue issues may be identified and surgical procedures to address them are performed. Once the patient, prosthodontist, and surgeon are satisfied with the prosthetic's function, soft tissue health, esthetics, and occlusion, it is removed. The prosthesis is then sent back to the dental laboratory where it is 3-dimensionally

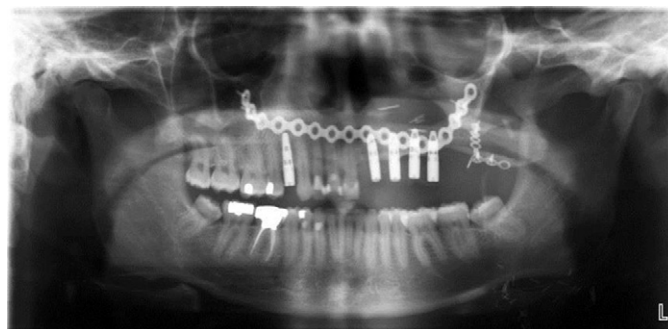


Fig. 37 Panoramic radiograph of implants placed into fibula. Same patient as in Figs. 15 and 16.



Fig. 38 Example of poorly cleansable, elongated buccal flanges on multiple implant supported fixed dental prosthesis.

laser scanned and a new CAD/CAM-milled, zirconium-based, screw-retained final prosthetic is manufactured. The advantage to this process, albeit expensive, is that it produces an extremely durable and retrievable replica of the temporary prosthesis, thus minimizing any further need for prosthetic or surgical interventions.

Secondary issues in the dentoalveolar reconstruction phase

During the prosthetic rehabilitation phase, 2 main issues have been noted in the authors' practice: the unfavorable biomechanics due to the implant-to-crown ratio and peri-implant soft tissue maintenance. In many composite wounds, the vertical discrepancy between the reconstructed bone and native bone leaves an elevated prosthetic platform, which led to this unfavorable ratio. Multiple implants splinted together are a requirement for long-term stability in these situations (see Fig. 27).

Implant hygiene is also difficult due to this discrepancy and can be made more difficult because of the prosthesis design. Excessive length of the prosthetic's buccal and lingual flanges on a nonremovable prosthesis greatly increases the difficulty for the patient and practitioner in visualizing and accessing the implant tissue interface (Fig. 38). The frequent lack of suitable peri-implant tissue combined with poor hygiene often leads to soft tissue inflammation with progressive implant thread exposure, bone loss, peri-implantitis, and implant loss (Fig. 39). Because of these issues, patients frequently undergo secondary procedures during the temporary prosthesis phase, such as



Fig. 39 Tissue inflammation surrounding implant-tissue interface with prosthesis removed. Reduction of prosthetic flanges and palatal grafting procedures were used to prevent this reaction.



Fig. 40 CAD/CAM design and manufactured, zirconium-based, multi-implant supported fixed dental prosthesis with cleansable, low-profile flanges and elevated framework for soft improved tissue cleansability.

a vestibuloplasty and palatal, allograft, or autogenous skin grafting procedures to address these peri-implant issues in an attempt to restore a more hygienic anatomy (see Fig. 21). The maxillofacial prosthodontist will also frequently modify the prosthesis by reducing the length of prosthetic flanges and elevate the prosthetic framework from underlying soft tissue to improve access for hygiene (Fig. 40).

Summary

Reconstruction of maxillofacial composite defects is a technically demanding and time-demanding process. It also requires a prolonged treatment course, a team approach, and meticulous planning that is prosthetic and esthetically driven. The use of vascularized flap reconstruction, dental implants, and computer-aided technology and advances in maxillofacial prosthetics has contributed immensely toward the goal of fully reconstructing victims of large avulsive wounds. Further advances in technology, surgical training, and maxillofacial prosthodontics will undoubtedly aid in minimizing the number of surgical interventions and maximize the final functional and esthetic results of these patients.

Acknowledgment

We appreciate the assistance of Greg Waskewicz CAPT, USN, DC for his clinical photographs and maxillofacial prosthetics contributions.

Further readings

- Baumann A, Schicho K, Klug C, et al. Computer-assisted navigational surgery in oral and maxillofacial surgery. *Atlas Oral Maxillofac Surg Clin North Am* 2005;13:41–9.
- Bui TG, Bell RB, Dierks EJ. Technological advances in the treatment of facial trauma. *Atlas Oral Maxillofac Surg Clin North Am* 2012;20:81–94.
- Cordova SW, Bailey JS, Terezides AG. Pectoralis major myocutaneous flap reconstruction of the mandible. *Atlas Oral Maxillofac Surg Clin North Am* 2006;14:171–8.
- Fernandes R. Fibula free flap in mandibular reconstruction. *Atlas Oral Maxillofac Surg Clin North Am* 2006;14:143–50.
- Futran ND, Farwell DG, Smith RB, et al. Definitive management of severe facial trauma utilizing free tissue transfer. *Otolaryngol Head Neck Surg* 2005;132(1):75–85.
- Futran ND. Maxillofacial trauma. *Facial Plast Surg Clin North Am* 2009;17:239–51.

Kademani D, Keller E. Iliac crest grafting for mandibular reconstruction. *Atlas Oral Maxillofac Surg Clin North Am* 2006;14:161–70.

Patel A, Levine J, Brecht L, et al. Digital technologies in mandibular pathology and reconstruction. *Atlas Oral Maxillofac Surg Clin North Am* 2012;20:95–106.

Powers DB, Will MJ, Bourgeois SL, et al. Maxillofacial trauma treatment protocol. *Oral Maxillofac Surg Clin North Am* 2005;17:341–55.

Xia JJ, Gateno J, Teichgraeber JF. Three-dimensional computer-aided surgical simulation for maxillofacial surgery. *Atlas Oral Maxillofac Surg Clin North Am* 2005;13:25–39.